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GENERAL PRACTICE SERIES

IMMUNIZATION

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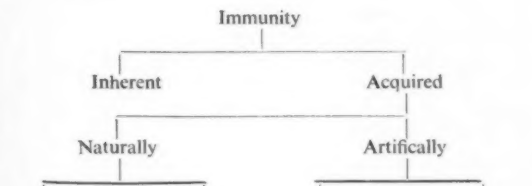
'Prevention is better than cure.' This is certainly so when immunization is safe, practicable and effective. Unfortunately it is not always so, and problems of various sorts arise. These are best solved by the judicious application of certain general rules based on the standard principles of immunity which will be reviewed briefly in this paper. Immunization will then be considered as it applies to different diseases and finally an attempt will be made to draw up a schedule for preventive inoculation.

PRINCIPLES OF IMMUNITY

It is well known, that resistance to infection may be *inherent* or *acquired*. Inherent immunity is genetically determined and needs no further comment. Acquired immunity can be so acquired either *naturally and actively* i.e. by having the disease, or alternatively *naturally and passively* as in infants who seem to be immune to measles, diphtheria, scarlet fever and chicken-pox for the first few months of life. These ways of acquiring resistance are best illustrated by the well-known schema:



Clearly, active natural immunity lasts for a longer period than passive immunity; it may also follow sub-clinical infection, as in those of us who have not had scarlet fever or diphtheria yet are immune to them. There are undoubtedly occasional breaches of the protection and the same disease, such as measles or diphtheria, occasionally occurs twice in an individual.

Acquired artificial immunity is 'introduced immunity' and is *passive* where antibodies are formed 'outside' the patient, e.g. in other humans or in horses. It is *active* when a vaccine containing modified or attenuated organisms or viruses or altered toxin is introduced and the patient manufactures his own antibodies; usually 2 or more injections are needed to induce a sufficient degree of immunity, because the primary stimulus merely prepares the way for more active response to the second stimulus. This immunity takes some weeks to develop but persists much longer than the passive type. As it wanes, it can be quickly raised again by a small 'booster' dose, the so-called 'recall dose' of the stimulating antigen.

DIPHTHERIA

Active immunization of infants with the prophylactic injection of diphtheria toxoid should be given at about 6 months of age. There are practically no reactions in children or infants with the use of the toxoids at present available, and failure to immunize is not only an unforgiveable omission, it is also a reflection of the fact that the public health authority concerned is not checking-up on immunization in its community.

For active immunization the preparations commonly used are APT and PTAP for children and TAF for adults. APT (Alum precipitated toxoid) or PTAP (Purified Toxoid Aluminium Precipitated) are given in 2 doses of 0·5 c.c. each with a 4-6 weeks interval. It is important to use 0·5 c.c. and 0·5 c.c. under 10 years, and 0·25 c.c. followed by 0·5 c.c. in older children. Injections should be given in opposite arms. The primary immunization is done before 1 year of age and booster doses of the same size are given at 3 years, 6 years and 10 years. For adults and children over 10 years TAF (Toxoid-antitoxin floccules) is advisable, since the others may cause pyrexial reactions. Dosage is usually 2 injections intramuscularly, at intervals of 4 weeks.

In certain persons—nurses working in fever hospitals, medical students etc.—it may be necessary to decide whether to immunize or not, and here the Schick test is of great value. A positive result indicates susceptibility to diphtheria and calls for immunization.

Passive immunization is used in treatment, antitoxin being given in varying doses according to the clinical type of the disease; anything from 20,000–100,000 units may be required.

To protect contacts, 1,000–5,000 units will protect for about 14 days and Schick-positive contacts should be actively immunized at the same time. It is well known that the administration of antitoxin should not be delayed while awaiting the result of bacteriological investigation.

Unfortunately, with antitoxin as with other sera there exists the risks of anaphylaxis and serum reactions, which are further discussed below.

WHOOPING COUGH

There is some doubt regarding the efficacy of vaccination against pertussis, but the weight of evidence favours the use of whooping-cough vaccine and active immunization against that disease. The Whooping-Cough Immunization Council of Great Britain (1949) found the incidence and severity of whooping cough in vaccinated groups to be less than in control groups. All infants should be given the vaccine, preferably before the age of 6 months, combined with the anti-diphtheria toxoid and tetanus toxoid (see below).

TETANUS

The high incidence of tetanus in South Africa makes immunization against this disease essential in all children. Active protection is afforded by tetanus toxoid given in 3 doses of 1 c.c. each (this may vary according to the preparation used), with an interval of 6 weeks between the 1st. and 2nd. This can be extended to 6 months, but no protection occurs until some weeks after the 2nd injection. A 3rd dose should be given 6-12 months after the 2nd and produces a level of protection sufficient for about 3 years. Thereafter, single booster doses of toxoid produce rapid increase in antibodies and such doses can be used if a suspicious injury is sustained. This avoids the repetition of antitoxic serum with the attendant danger of anaphylaxis. It is well known that the United States Army during the last war actively immunized personnel against tetanus and then treated each injury with toxoid (and not antitoxin). This afforded excellent protection, as the negligible incidence of tetanus showed, and the plan can safely be extended to civilian practice and particularly in our country. We shall thus reduce the incidence of allergic reactions and lessen the danger of anaphylaxis. Unfortunately, at present, antitoxin must be used when wounds are received and there are no hard and fast rules regarding the type of wound in which antitoxin should be administered. Some casualty departments give antitoxin to every casualty. In general, puncture wounds or lacerated wounds containing dead tissue or foreign bodies call for immunization, and also wounds contaminated with soil or manure. Also frequently forgotten but requiring protection is the unhygienically-induced abortion and the septic burn; tetanus following these is not rare and unfortunately has a very high mortality.

As things are, we must give 1,500–3,000 units of antitetanic serum ATS to injured patients but, looking to the future, we should immunize those we can with toxoid.

Two questions are frequently asked:

1. What to do if a patient is serum-sensitive or allergic and a wound is sustained? In my opinion, ATS need not be given when the danger of anaphylaxis is great, because adequate cleaning and careful follow-up dressing is usually protection enough.

2. Is it too late to actively immunize a patient at the time his wound is received? In my opinion, all persons should be actively immunized when injured, particularly children given to walking barefoot, though antitoxin must be given at the same time. Do not omit following-up with the 2nd and the 3rd toxoid injections to complete the active immunization.

COMBINED DIPHTHERIA-PERTUSSIS-TETANUS

This is the most convenient method of immunization and therefore the commonest used, but it is worth noting that tetanus toxoid can be combined very effectively with anti-typhoid vaccine TAB, and it may be easier to immunize against diphtheria-pertussis in the first year and tetanus-typhoid in the second or third. However, most practitioners are content to use the diphtheria-pertussis-tetanus antigen, of which several varieties are available. Usually 3 doses of 0·5 c.c. each are required to be given intramuscularly at 4-6 weeks intervals, and a booster dose is advisable 2 years later. The level of protection against tetanus is not always adequate with the mixed toxoid and if a contaminated wound is sustained, prophylactic tetanus toxoid should be given separately.

It is important to know that toxoids will not produce reactions in serum-sensitive persons.

MEASLES

Some believe it is sensible to allow the disease to be contracted when circumstances are favourable and the institution of 'measles tea-parties' has been seriously advised in children about 5 years old. This may be a drastic step, but in a household it is often advantageous to let the children have measles together, if possible. Active immunization is not yet practicable. For passive immunization, to protect exposed children who have not had the disease, gamma globulin can be used. This material constitutes approximately 11% of all plasma protein and with few exceptions contains all the blood antibodies. A Medical Research Council (1948) sub-committee substantiated the manufacturers' claims for the reagent and doses of 0·1-0·2 c.c. per lb. of body weight, given in the first 5 days of exposure may protect against measles. If given up to the 10th day of exposure it may attentuate the disease.

RUBELLA

A pregnant woman in the first two to three months may, if she develops rubella, give birth to a child with serious congenital defects such as deafness or blindness. The only protection in such a woman exposed to the disease is the injection of a large dose of gamma globulin prepared from the serum of convalescent cases of rubella.

ENTERIC FEVER

Active immunization against the typhoid-paratyphoid group is essential in South Africa and is best carried out at about

6 years of age. Thereafter booster doses can be given every 2 or 3 years, particularly in those areas where the disease is prevalent. The usual method is to use TAB vaccine containing killed cultures of the organisms. The preparation issued by the S.A.I.M.R. requires 2 injections of 0·5 c.c. or 1 c.c. (according to the strength), the two to be given with an interval of 10-14 days.

SCARLET FEVER

Active immunization is rarely used today and chemotherapy is the preferred method of preventing spread of this disease.

VACCINATION

The virus of vaccinia does not occur naturally but is a 'laboratory' strain produced by animal passage, usually through the cow, monkey or rabbit, of the virus of smallpox or cowpox. These viruses are related in that the immunogenic complexes are identical, so that vaccinia virus protects against smallpox, i.e. variola major and variola minor (known as alastrim or kaffir-pox).

It is important that the lymph used be fresh and it is best stored under refrigeration. Unfortunately for technical reasons dried lymph is not manufactured.

It is surprising how few doctors here practice the multiple-pressure method (or acupuncture) which is the method of choice in the United States of America and has been officially recommended in Great Britain. A drop of lymph expelled from its capillary container by means of a rubber ejector (*not* by blowing it out) is placed on the clean, dry skin; through it, with the *side* of the point of a straight needle, which should be flat-sided or triangular in section, held *parallel to the skin surface* and moved rapidly up and down at right angles to this plane, a number of pressures are made with a force just sufficient to demonstrate the elasticity of the skin. About 30 pressures at any one site are sufficient and even 10 or 20 may be effective especially in primary vaccinations. Excess lymph may then be removed with a sterile swab and the site left un-dressed, or a small dry gauze may be applied with a thin layer of strapping.

The scratch technique ('linear insertion') is often used and it is well to recall that the scratch should be superficial and insufficient to cause free bleeding. The site must be left to dry and a dry dressing applied.

Three types of reaction may occur in vaccination:

1. *The primary reaction.* This is successful vaccination and produces a papule at the 4th-7th day. Then follows vesiculation and local inflammation, a pustule forms and finally a scab.

2. *The accelerated reaction* ('*vaccinoid*'). This is successful vaccination in a subject already partially immunized, and response is modified in proportion to the degree of resistance already present. The intensity of the local reaction is lessened, with a small or evanescent vesicle, often present on the second day.

3. *The immediate reaction.* A red itchy papule develops within 12 hours, and reaches its height within 24-72 hours and then fades. This is to be regarded as an unsuccessful vaccination frequently occurring with inactivated lymph, and *not* as a reaction of immunity. The vaccination should then be repeated.

In patients with skin diseases there exists a danger of generalization of the virus by inoculation. Eczema, especially in children, burns, and open wounds, should be regarded as contraindications. Vaccination should also not be undertaken during the course of any acute infection; nor is it advisable to combine it with any other virus inoculation. Because of the nature of the viruses involved, if yellow-fever vaccine is also to be administered it should be given not less than 4 days before or 3 weeks after primary vaccination.

The complications of vaccination are non-specific sensitivity rashes, mainly of the erythema multiforme type, generalization of the disease by haematogenous spread (exceedingly rare), accidental spread by inoculation, and post-vaccinial encephalitis (which also appears to be rare in South Africa, though relatively common in some parts of Europe).

ANTERIOR POLIOMYELITIS

Active immunization against this disease is now a reality and the efficacy of the vaccine is no longer in doubt. The subject has been fully ventillated in this *Journal.*

Under certain circumstances immunization may be required against *yellow fever, typhus, plague* and *cholera*; this is carried out at central stations or laboratories controlled by the Union Department of Health, or by arrangement with the Department.

SERUM REACTIONS

It is wise to carry out a sensitivity test before administering serum. For this 0·2 c.c. of a 1-in-10 dilution of the preparation is injected subcutaneously. Intradermal tests are unreliable and reveal dermal sensitivity, which does not necessarily parallel general sensitivity or anaphylaxis. The subcutaneous route is followed in persons suspected of being sensitive, the dose being increased tenfold at 2-hourly intervals. After 4 or 5 injections without reaction, the full dose can be given intramuscularly. Adrenaline ampoules and a syringe must always be in readiness when serum is given.

SCHEDULE FOR IMMUNIZATION

A. *Babies during first year.* 1. Diphtheria-Whooping-cough-Tetanus Combined Method. Injections of 0·5 c.c., 1 c.c. and 1 c.c. at one month intervals starting at 2-4 months.

2. Uncombined Method. Separate injections of Diphtheria toxoid (APT or PAPT) 0·5 c.c. and 1 c.c. at one month interval and Pertussis vaccine.

3. Vaccination against Smallpox.

4. Immunization against Poliomyelitis.

B. *At two years.* Booster dose against Diphtheria-Whooping-cough-Tetanus.

C. *At school age.* 1. Repeat booster doses of Diphtheria-Whooping-cough-Tetanus.

2. Repeat vaccination against Smallpox and thereafter every 5 years.

3. Immunization against Enteric (TAB).

D. *Adults.* 1. Vaccination against Smallpox.

2. Typhoid repeat every 3 years.

3. Tetanus toxoid. In people exposed to injuries of the dangerous sort, e.g. farm labourers etc., booster dose of Tetanus toxoid every 2 years.

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EDITORIAL

FIRST EXAMINATIONS OF THE COLLEGE OF PHYSICIANS AND SURGEONS OF SOUTH AFRICA

The College of Physicians and Surgeons of South Africa has announced its intention of holding its first postgraduate examinations in October 1957. The examinations which will afford successful candidates entry to the College are the Primary examination for the Fellowship of the College of Surgeons (South Africa), the Fellowship of the College of Physicians (South Africa), and the Fellowship of the College of Obstetricians and Gynaecologists (South Africa).

At its meeting in March 1957 the South African Medical and Dental Council approved in principle that these three higher qualifications be recognized as 'additional qualifications'. At the time that this matter was before the South African Medical and Dental Council, the exact titles of all the qualifications had not been settled, and therefore it was not possible for a formal recommendation to be made to the Minister of Health that the higher qualifications be gazetted as additional qualifications. The titles, as announced elsewhere in this issue, (pp. xxiv and xxv), have now been decided upon by the Council of the College.

It must be clearly understood that the qualifications of the College have in principle been accepted as '*additional*' qualifications. The question of their recommendation by the South African Medical and Dental Council for acceptance and gazetting by the Minister as *higher* qualifications for purposes of admission to the statutory specialist register of the South African Medical and Dental Council is still under discussion by the appropriate committees of the Medical Council.

The first Primary examination for the F.C.S. (S.A.) will be attended by Sir Harry Platt, President of the Royal College of Surgeons of England, and it is hoped that the first examination for the Fellowship of the College of Physicians (S.A.) will be attended by Sir Russell Brain, President of the Royal College of Physicians of London. The examiners will be drawn from a panel already compiled by the Council of the College, and will include examiners with experience of the standard of higher qualifications of South African Universities which are already recognized as higher qualifications for the purposes of the specialist register, and with standards of overseas examinations of equal calibre.

It is difficult to set examination standards except by holding these examinations and permitting the standard to be studied. The standard of the Primary F.C.S. (S.A.) will be such as will be acceptable to the Royal College of Surgeons of Edinburgh and England as exempting from the Primary examinations for the Fellowships of these two Royal Colleges. Any doubts that may exist in the minds of prospective candidates for the Primary F.C.S. (S.A.) regarding the ultimate acceptance of the Final F.C.S. (S.A.) by the South African Medical and Dental Council as a higher qualification in terms of the specialist regulations will be allayed by the knowledge that they, having completed the Primary F.C.S. (S.A.), will at least be able to proceed overseas to take the Final examinations of the Royal College of Surgeons of England or Edinburgh, both of which are registrable as higher qualifications in terms of the South African specialist regulations.

There is no primary for the F.C.P. (S.A.) and the F.C.O.G. (S.A.). The Council of the College has nevertheless, by announcing the holding of these two examinations, afforded the opportunity for prospective candidates to enter the examinations in the faith that these qualifications will in the future be registrable with the South African Medical and Dental Council as additional qualifications and, it is hoped, as higher qualifications as well.

The first examinations will be held in Cape Town. Future College examinations will be held in different centres in South Africa. Cape Town has been selected for the first examinations for the main reason that the University of Cape Town has offered courses of instruction in Anatomy, Physiology and Pathology for candidates for the Primary F.C.S. (S.A.) in preparation for the examination.

Although this is not strictly relevant to the subject of its examinations, the College Council has announced that the Registrar of Companies has approved the inclusion in the College of Physicians and Surgeons of South Africa of a division of Obstetrics and Gynaecology equal in standing with the divisions of Surgery and Medicine. Furthermore, the Council of the College will place before the second Annual General Meeting of the College in September 1957 recommendations concerning the title of the College, with a view either to including in it not only 'Physicians and Surgeons' but also 'Obstetricians and/or Gynaecologists' or, alternatively, to have a more comprehensive and brief title which will cover the three major branches of Medicine, Surgery, and Obstetrics and Gynaecology, without specifically naming any one.

The announcement of the College examinations is an expression of the College's faith in its own purposes, and the support which the examinations will in future be given will be an indication that one of its important purposes has been effected.

VAN DIE REDAKSIE

DIE NA-FLEBITIESE LEDEMAAT

Die pasiënt wat aan na-flebitiese gevolge ly is dikwels liggaamlik ernstig ongeskik en bied 'n uiters moeilike terapeutiese probleem. Wederkerende edeem, ulserering, dermatitis, oppervlakkige spataartjies, weefselontsteking, elefantiase en fibrose, tesame met subjektiewe simptome soos bv. vermoeidheid, swaarheid en pyn, mag 'n ondraaglike las op die pasiënt, haar familie en haar werkgewers lê. Die hoofprobleem is die verhoogde druk in die are wat volg op die herkanalisering van die getromboseerde diepliggende are. Die kleppe wat by die trombus vasgevang is, word onherroeplik verniel en met die styging in aardruk, is die oppervlakkige are geneig om te verwyd en klepvliesondoeltreffendheid te ondergaan. Die aardruk kan gemeet word en siekte van die diepliggende are deur flebografiese bestudering bevestig word. Diodone word in 'n aar op die rugkant van die voet ingespuit en, met die aarterugvloei deur 'n aar-afbinder versper bokant die knie, word goeie beelde verkry. Brewer[1] het aan die hand gedoen dat arterioveneuse koppelings, na trombose van die diepliggende are, oopgaan en dat hierdie koppelings van groot belang by die voortbrenging van spatare is. Ruim 70-80% van aarsere is na-tromboties en slegs ongeveer 20% is as gevolg van spatare. Cockett[2] het die veelvuldigheid waarmee sere op die laer derde van die mediale kant van die been voorkom, verklaar op die basis van 3 konstante are wat die diepliggende seningvlies perforeer en direk in die diepliggende are dreineer. Ulserering word deur klepvliesondoeltreffendheid en verhoogde druk in hierdie are veroorsaak, en om aarsere by hierdie posisie te behandel, raai hy aan dat uitsnyding van die seer en buite-fasiale afbinding van die onbekwame perforerende are gedoen word. Anning[3] het daarop gedui dat hierdie are slegs ondoeltreffend word wanneer daar 'n gebrek in die diepliggende stelsel is wat drukverhoging veroorsaak, en Cockett se operasie is nutteloos om dit op te klaar. Die onmiddellike resultate van Cockett se operasie was goed, maar dieselfde is waar van baie ander operatiewe metodes vir hierdie toestand wat nie hulle vroeë belofte gehandhaaf het nie. Dit moet nog bewys word of die lang-termyn resultate die entoesiastiese aanname van Cockett se sienswyses regverdig.

'n Groot aantal operatiewe metodes is aanbeveel vir die behandeling van die na-flebitiese ledemaat; afbinding van die grotere diepliggende are, uitsnyding van oppervlakkige spataartjies, inspuitingsterapie, lumbale simpatektomie en, onlangs, deur Eisman en Malette,[4] die konstruksie van nuwe kleppe. Hierdie operasies is òf op hulle eie, òf in verskeie permutasies en kombinasies gedoen. Die blote bestaan van so baie verskillende metodes is op sigself veelseggende bewys van die algemene onbevredigende resultate wat met die behandeling saamgaan. In 1948 het Bauer[5] afbinding van die kniekuil-are, wat eerstens deur Parona gebruik is, weer ingestel. Die logiese rede vir die operasie is dat, deur terugvloeiing te stuit, word aardruk verminder en die bloed deur spierwerking *via* die kollaterale are gepomp. Boyd, Catchpole en Jepson[6] het 'n reeks van 42 pasiënte, wat 'n groot aar-afbinding vir trombose van die diepliggende are gehad het, opgevolg. By geeneen van die gevalle was die na-operatiewe aardruk laer as voor die operasie nie.

EDITORIAL

THE POST-PHLEBITIC LIMB

The patient suffering from post-phlebitic sequelae is often severely disabled, and presents a most difficult therapeutic problem. Recurrent oedema, ulceration, dermatitis, superficial varicosities, cellulitis, elephantiasis and fibrosis, together with subjective symptoms such as fatigue, heaviness and pain, may impose an intolerable burden on the patient, her family, and her employers. The essential problem is the venous hypertension which results when the thrombosed deep veins recanalize. The valves caught up in the thrombus are hopelessly destroyed and, with the rise in venous pressure, the superficial veins tend to dilate and suffer valvular incompetence. The venous pressure can be measured, and disease of the deep veins confirmed by phlebographic study. Diodone is injected into a vein on the dorsum of the foot and, with the venous return obstructed by a tourniquet above the knee, good pictures are obtained. Brewer[1] has suggested that following deep-vein thrombosis arteriovenous shunts become opened, and that these shunts are of great importance in the production of varicose veins. Fully 70-80% of venous ulcers are post-thrombotic, and only about 20% are varicose. Cockett[2] has explained the frequency with which ulcers occur on the lower third of the medial side of the leg on the basis of 3 constant veins which perforate the deep fascia and drain directly into the deep veins. Ulceration is caused by valvular incompetence and increased pressure in these veins, and to deal with venous ulcers in this situation he advises excision of the ulcer and extra-fascial ligation of the incompetent perforating veins. Anning[3] has pointed out that these veins only become incompetent when there is a defect in the deep system causing hypertension, and Cockett's operation does nothing to correct this. The immediate results of Cockett's operation have been good, but this is also true of a good many other operative procedures for this condition which have not maintained their early promise. It is yet to be seen whether the long-term results will justify the enthusiastic acceptance of Cockett's views.

A great many operative procedures have been advocated in the treatment of the post-phlebitic limb; major deep-vein ligation, excision of superficial varicosities, injection therapy, lumbar sympathectomy and, recently, by Eisman and Malette,[4] the construction of new valves. These operations have been performed either alone or in various permutations and combinations. The very existence of so many different procedures is in itself eloquent testimony to the general unsatisfactory results attending the surgery. In 1948 Bauer[5] re-introduced popliteal-vein ligation, first employed by Parona. The rationale of the operation is that, by halting retrograde flow, venous pressure is reduced, and the blood is pumped by muscular action *via* the col-

Deur middel van flebografie het hulle gevind dat, binne 'n paar maande na die aar verdeel is, ondoeltreffende, kleplose kollaterale op die plek van afbinding ontwikkel het. Dit is dus duidelik dat dit onwaarskynlik is dat groot aaronderbreking die hemodinamiese doeltreffendheid van die ledemaat verbeter.

Linton,[7] 'n erkende outoriteit op hierdie gebied, glo dat by die behandeling van die na-flebitiese ledemaat, 'n onderskeid gemaak moet word tussen die ledemaat met ulserering en die ledemaat daarsonder. Waar daar geen ulserering is nie, raai hy 'n wesenlike konserwatiewe stelsel aan; wanneer daar 'n seer aanwesig is, pas hy radikale chirurgie toe. Linton se operasie is inderdaad baie radikaal en bestaan uit 5 stappe: (1) Die oppervlakkige are, beide lank en kort, word verwyder. (2) Die diepliggende aarstelsel word onderbreek deur afbinding van òf die oppervlakkige femorale aar net distaal van die profunda, òf van die kniekuil-are by die knie. (3) Die verbindingsare tussen die oppervlakkige en diepliggende stelsels onderkant die knie word deur 'n vertikale snit aan die binnekant van die laer been onderbreek. (4) Deur dieselfde opening word die diepliggende seningvlies gesny in 'n poging om die limfdreinering van die vel en onderhuidse weefsels te verbeter. (5) Die pasiënte word beveel om vir 'n onbepaalde tyd 'n rekbare ondersteuning van die tone tot net onderkant die knie te dra.

Owens en Anderson,[8] wat ondervinding van die meerderheid van die beter-bekende chirurgiese metodes gehad het en ontevrede gevoel het met die resultate wat verkry was, het 'n deeglike behoudende terapeutiese metode begin. Hulle het hul stelsel gebaseer op die prinsiep van progressiewe ambulatoriese behandeling, wat eerstens deur John Homan in 1917 gebruik is. Die hoof grondslag van behoudende behandeling is elevasie, samepersing, en oefening van die eind-ledemate, tesame met beheer van ontsteking en aandag aan alle verwante ongesteldhede. Dit is belangrik dat die samewerking van die pasiënt verkry word en daar word gouste in hierdie doel geslaag deur 'n eenvoudige verduideliking van die psiologiese en patologiese veranderinge wat die toestand veroorsaak. Een van die hoofdoele van behandeling is om die spierstelsel van die kuit op te bou, aangesien hoë aardruk met normale funksie verenigbaar is, mits die pompwerking van die kuitspiere doeltreffend is.[9] Met behoudende metodes was hulle resultate 85% suksesvol en chirurgie is slegs gebruik om indolente sere uit te sny en oor te plant. Hulle dring daarop aan dat die eind-ledemate edeem-vry moet wees voordat ambulasie toegelaat word, sodat die kollaterale die beste kans het om te ontwikkel.

Ongelukkig is daar gewoonlik sosio-ekonomiese struikelblokke wat die weg tot behoudende behandelingsmetodes versper. Baie pasiënte, by beskouing van hulle opgeswelde, verkleurde, ulsererende en pynlike bene, sal met blydskap die genoeglike vooruitsig van rustige elevasie van daardie moeë bene vir 'n kwartier verskeie kere per dag verwelkom, maar hulle kla dat omstandighede dit onmoontlik maak. En hier kom ons teenoor die hooffaktor van ons probleem te staan.

1. Brewer, A. C. (1950): Brit. Med. J., **2**, 270.
2. Cockett, F. B. (1955): Brit. J. Surg., **43**, 260.
3. Anning, S. T. (1956): Post. Med. J., **32**, 585.
4. Eisman, B. en Malette, W. (1953): Surg. Gynec. Obstet., **97**, 731.
5. Bauer, G. (1948): J. Int. Chir., **8**, 937.

lateral veins. Boyd, Catchpole and Jepson[6] have followed a series of 42 patients who had a major vein ligation for deep-vein thrombosis. In none of the cases was the post-operative venous pressures lower than before the operation. By means of phlebography they found that, within months of the vein being divided, incompetent valveless collaterals developed at the site of the ligation. It is clear then that major vein interruption is unlikely to improve the haemodynamic efficiency of the limb.

Linton,[7] an acknowledged authority in this field, believes that in the treatment of the post-phlebitic limb a distinction must be made between the limb with ulceration and the limb without. Where there is no ulceration, he advises an essentially convervative regime; in the presence of an ulcer, he practises radical surgery. Linton's operation is very radical indeed, and consists of 5 steps: (1) The superficial veins, both long and short, are removed. (2) The deep venous system is interrupted either by ligating the superficial femoral vein just distal to the profunda or the popliteal veins at the knee. (3) The communicating veins between the superficial and deep systems below the knee are interrupted through a vertical incision on the inner side of the lower leg. (4) Through the same incision the deep fascia is incised in an attempt to improve the lymphatic drainage of the skin and subcutaneous tissues. (5) The patients are instructed to wear elastic supports for an indeterminate period from the toes to just below the knee.

Owens and Anderson,[8] having had experience with most of the better-known surgical procedures and feeling dissatisfied with the results obtained, initiated a thoroughly conservative plan of therapy. They based their regime on the principle of progressive ambulatory treatment, first used by John Homan in 1917. The essential basis of conservative treatment is elevation, compression, and exercise of the extremities, together with control of infection and attention to any allied disorders. It is important to secure the cooperation of the patient, and this is best achieved by a simple explanation of the physiological and pathological changes underlying the condition. One of the major aims of treatment is to build up the musculature of the calf, since high venous pressures are compatible with normal function provided the pumping action of the calf muscles is efficient.[9] They obtained 85% successful results by conservative methods, and surgery was used only to excise and graft indolent ulcers. They insist that the extremities must be oedema-free before ambulation is allowed so that the collaterals have the best chance of developing.

Unfortunately there are commonly socio-economic obstacles to conservative methods of treatment. Many patients, miserably contemplating their swollen, discoloured, ulcerated and painful legs, would joyfully welcome the delightful prospect of restful elevation of those weary legs for a quarter of an hour several times a day; but they protest that circumstances make it impossible. And here we come up against the main factor in our problem.

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RIFT VALLEY FEVER IN A WILD FIELD RAT (ARVICANTHIS ABYSSINICUS): A POSSIBLE NATURAL HOST

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Wherever a virus of the arthropod-borne group occurs it is of interest to know in which species of the local vertebrate fauna the agent is able to cause an infection during the course of which there is a viraemia. It is known that an agent which, if not identical, is very closely related to Rift Valley Fever virus (RVF), occurs in the area in which this laboratory is situated[1]. It is also known that neutralizing antibodies to RVF virus may be found in the blood of wild caught rats, *Arvicanthis abyssinicus nubilans* Wroughton, from the same area.[2] It is the purpose of this communication to report the results of some studies on the circulation of RVF virus in a recently colonized strain of *A.a.nubilans* which is now maintained in this laboratory.

MATERIALS AND METHODS

Virus. This was a strain of RVF virus isolated from mosquitoes, *Eretmapodites* spp., in Bwamba County, Uganda.[3] The stock consisted of lyophilized whole mouse serum taken from animals dead or moribund after inoculation of material which had been through about 160 mouse passages. It was stored in flame-sealed glass ampoules at −25°C in a mechanical deep freeze.

Rats. Arvicanthis abyssinicus nubilans (hereafter referred to as '*Arvicanthis*' or 'rats') from a colony started in October 1955, with 4 pairs of wild caught animals. These were taken in traps on the Entebbe peninsula. At first some difficulty was experienced in inducing these animals to breed but after they were supplied with 6-inch lengths of ceramic drain-pipe to serve as nesting boxes and provided with a diet rich in greenstuff, breeding began. After 4 or 5 generations the drain pipes were no longer necessary and the strain now breeds freely as long as an adequate diet is maintained.

Diluent. This was a 0·75% suspension of Armour's Fraction-V bovine plasma-albumin in phosphate-buffered saline at pH 7·4. The suspension was sterilized by Seitz filtration. This is referred to as BPA.

Mice. Swiss albino mice from the Institute colony were used. This was originally stocked with animals bought from Carworth Farms, New York, USA.

Titrations for Infectivity. These were carried out with serial 10-fold dilutions of infective material, either serum or a 10% suspension of tissue in BPA, which were inoculated intracerebrally (0·03 ml. per mouse) into groups of 5 adult mice. The 50% end-point was calculated by the methods of Reed and Muench[4] from deaths occurring between the 2nd and 5th days after inoculation.

Protection Tests. These were carried out by mixing equal parts of the material to be tested, and virus suspension diluted to such a point that the serum-virus mixture was estimated to contain approximately 100 mouse-LD_{50} per 0·06 ml. of total volume. The mixtures were well shaken and incubated for 1 hour at 37°C before being inoculated intraperitoneally (0·06 ml. per mouse) into groups of 5 mice. Each test was controlled by a titration of the challenging virus in BPA and another in known RVF immune serum.

Interpretation of Protection-Test Results. In a group of 5 mice, if all, or all but one, of the mice survived, the result was accepted as being positive. If only 1 or 2 mice survived the test was deemed negative. All other combinations were considered inconclusive. In a group of 4 mice, if all lived it was considered positive and if all died negative; any other combination was deemed to be inconclusive. Groups of less than 4 mice were not accepted. Sera which gave an inconclusive result were not retested, because circumstances did not always permit the re-bleeding of the animals and, for the purpose of these experiments the recording of an inconclusive test does not affect the final results.

Collection of Blood Specimens. The rats were anaesthetized with ether and were bled from the heart by means of a saline-wetted 0·5-ml. all-glass tuberculin-type syringe fitted with a 5/8th-inch 25-gauge needle. When adult rats were bled, 0·2 ml. of blood was taken and mixed with 1·8 ml. of BPA. When infant rats were bled, 2 members of the litter were chosen and 0·1 ml. was taken from each; both these 0·1 aliquots were added to the same tube containing 1·8 ml. of BPA. The 10^{-1} dilutions of whole blood so obtained were allowed to stand at room temperature until clots formed, when they were transferred to a 4°C refrigerator. After some hours at 4°C (this period varied between 4 and 18 hours) the specimens were centrifuged at 2,000 r.p.m. for 7 minutes in an angle-head machine. The supernatant fluid was removed with a Pasteur pipette and transferred to a sterile tube, whence aliquots were removed for use.

Histological Technique. Fixation was carried out in Carnoy's fluid, after which the tissues were embedded in paraffin wax and sections cut on a rotary microtome. Staining was by Mayer's haematoxylin and eosin.

EXPERIMENTAL

A pilot experiment was set up in an attempt to discover the most suitable route of inoculation and the degree of susceptibility of *Arvicanthis* to RVF virus. In this experiment 5 groups, each of 5 rats, were inoculated intraperitoneally and received doses of 10^8, 10^6, 10^4, 10^3 and 10^2 mouse-LD_{50} per rat. Two further groups were included, one being inoculated intracerebrally and one subcutaneously; each rat in these two groups received 10^6 mouse-LD_{50}. None of the animals inoculated with 10^8 LD_{50} showed any sign of illness, irrespective of the route of inoculation. In the other groups which were inoculated intraperitoneally, of those which received 10^6 LD_{50}, 3 died; and 1 died in each of the 10^4 LD_{50} and 10^3 LD_{50} groups, while none died in the 10^2 LD_{50} group. The animal which died in the 10^3 LD_{50} group was found at the point of death during the checking period on the second day following inoculation. This animal was taken for autopsy and by the time that it reached the laboratory it had died. A portion of heart-blood and a portion of liver were taken for titration and liver was taken for histological study. A portion of the liver suspension was also used to inoculate a further group of *Arvicanthis* by the intraperitoneal route, each animal receiving $10^{2 \cdot 9}$ mouse-LD_{50}. None of them showed any sign of illness. The results of these titrations were unexpected in that the serum-titre was higher than that for the liver, being $10^{6 \cdot 5}$ and $10^{5 \cdot 1}$ mouse-LD_{50} per 0·03 ml. respectively. Histological sections prepared from the liver of the same animal showed changes consistent with an infection by RVF virus. There were no large areas of destruction or haemorrhage, but nuclear changes were widespread and included margination of pyknotic nuclear chromatin and the formation of eosinophilic intranuclear inclusion-bodies. A fair amount of cell debris was in evidence, presumably derived from those cells which had disintegrated as a result of the pathological process induced by the virus. There were some cells which had undergone an eosinophilic degeneration of the cytoplasm; in these, small densely basophilic nuclear fragments were always to be seen. There was no polymorphonuclear infiltration.

It was decided to follow up this experiment by a check on the immune status of the surviving animals. They were accordingly kept until 6 weeks had elapsed since the inoculation of the original groups. At this time all those animals which had not succumbed either to RVF or non-specific intercurrent illness, including the group to which a passage was made from the one rat that had died on the second day, were bled to provide material for a mouse protection test. By this time a number of the female animals had produced litters and where it was possible to identify the young of a particular female they were also bled and their sera were included in the test. In this protection test the control titrations indicated that the challenge was 63 mouse-LD_{50}.

The results of these tests are set out in Table I, from which it may be seen that of the 23 adult-rat sera tested, 14 gave positive results, 6 gave inconclusive results, and only 3 contained no detectable antibody. Of the 7 infant-rat sera tested, 6 gave positive results and one a negative result. With regard to the distribution of the 'inconclusive' results, 2 occurred in each of the 10^8 LD_{50} groups which were inoculated by the intraperitoneal and subcutaneous routes, though in both these cases one of the inconclusives was a female animal the infants of which gave a positive result. The other 2 inconclusive results occurred in the groups which received the smallest inocula and in which there were animals which gave negative results. One of these negative results was obtained from a female animal whose young failed to show any signs of neutralizing substances in the serum. The remaining negative result was given by the serum of a male animal inoculated with 10^4 LD_{50} by the intraperitoneal route. Also shown in Table I are details concerning the dose of virus administered and its route of inoculation; the number of animals in each group which died of RVF; and the number which died of unknown causes in the 6 weeks waiting-period. It may also be seen from the results in Table I that only 5 of the 35 rats in the original groups died of RVF and that, of these, 3 died following a dose of 10^6 mouse-LD_{50}, and 1 each after doses of 10^4 and 10^3 mouse-LD_{50}, all having been inoculated by the intraperitoneal route.

TABLE I. RESULTS OF PILOT EXPERIMENT

Route of Inoculation	*Mouse LD_{50} Administered*	*Rats Dead from RVF*	*Rats Dead Non-specific*	*Identification No. and Sex Survivors at 6 Weeks*	*P.T. Results*	
					Adults	*Infants*
IP	10^8	0	2	3 M 2 F † 1 F	Pos Inc Inc	Pos
	10^6	1 on day 2 2 on day 3	0	9 F 10 M	Pos Pos	Pos
	10^4	1 on day 2	2	5 F 4 M	Pos Neg	Pos
Passage	10^3	1 on day 2*	1	11 M 12 F 13 F	Pos Pos Pos	Pos
	10^2	0	3	14 M 15 F	Inc Neg	Neg
	$10^{2 \cdot 9}$	0	1	16 M 18 M 17 F 19 M	Pos Pos Inc Neg	
IC	10^8	0	2	6 F 7 M 8 M	Pos Pos Pos	Pos
SC	10^8	0	1	21 M 22 M 23 F † 20 M	Pos Pos Inc Inc	Pos

* Titrations in mice of blood and liver from this rat gave figures of $10^{5 \cdot 6}$ and $10^{6 \cdot 5}$ LD_{50} per ml. or per g. respectively.

† In these cases it may be seen that the infant rats gave a clean positive result whereas the mother rat gave an inconclusive result, suggesting that the inconclusive results should really have been read as positives.

Following the findings reported above it was decided to carry out a further experiment, but using animals which were known to be non-immune at the beginning of the experiment. To provide these known non-immunes, 54 rats were brought into the laboratory in separate cages and were immediately numbered. They were then bled and the 10^{-1} dilutions of the blood used to set up a protection test. In this instance the stock virus gave a 10-fold lower titration figure than expected and the challenge was 10 mouse-LD_{50} instead of the desired 100 mouse-LD_{50}. In spite of this low challenge none of the sera showed any sign of neutralizing power. Because this test was quite adequate to provide the information required, namely that none of the rats concerned had experienced an infection with RVF, it was deemed unnecessary to repeat it, though a challenge of this magnitude would not have been acceptable for a test which contained any 'positive' sera.

Eight groups, each of 5 known non-immune *Arvicanthis*,

all of which were kept in their individual boxes, were then inoculated intraperitoneally with successive 10-fold dilutions of stock virus, one dilution being used for each group. The control titration subsequently showed that the doses administered ranged from $10^{7\cdot6}$ down to $10^{0\cdot6}$ mouse-LD_{50}. Only one of the rats died after the administration of the virus. This was one of the animals that received a dose of $10^{7\cdot6}$ LD_{50} and which died while aborting a litter of immature foetuses on the second day after inoculation. At autopsy there was quite advanced post-mortem change in the organs, none of which was kept for histological study. A specimen of clotted heart-blood was taken and a 10% suspension was made of this in BPA containing penicillin and streptomycin. This was used to set up an identification test, which consisted of paired titrations in BPA and RVF immune rabbit serum. The figures obtained were $10^{4\cdot8}$ and $10^{2\cdot4}$ respectively and show that the rat died with circulating RVF virus in its blood.

Six weeks after the experiment just described was carried out, all the surviving *Arvicanthis* were bled and a protection test was set up with their sera. The results obtained in this test, which employed a challenging dose of 80 mouse-LD_{50}, are set out in Table II, together with details of the size of

TABLE II. RESULTS OF FIRST EXPERIMENT CARRIED OUT USING KNOWN NON-IMMUNE RATS

Inoculum in Mouse LD_{50}	*Ident. No. of Rats Alive at 6 weeks*	*Protection Test Result*	*Inoculum in Mouse LD_{50}*	*Ident. No. of Rats Alive at 6 weeks*	*Protection Test Result*
$10^{7\cdot6}$	24*	Pos	$10^{3\cdot6}$	47	Pos
	26	Pos		48	Pos
	27	Inc †		49	Pos
	28	Pos		50	Pos
				52	Pos
$10^{6\cdot6}$	30	Pos			
	31	Pos	$10^{2\cdot6}$	53	Pos
	32	Pos		54	Pos
	33	Pos		55	Pos
	36	Pos		57	Pos
$10^{5\cdot6}$	37	Pos	$10^{1\cdot6}$	58	Neg
	38	Pos		59	Pos
	40	Pos		60	Neg
				62	Inc
$10^{4\cdot6}$	42	Pos			
	43	Pos	$10^{0\cdot6}$	64	Neg
	44	Inc††		65	Neg
	45	Pos		66	Neg
	46	Pos		67	Neg

* Rat 25 died on 2nd post-inoculation day. Blood virus titre $10^{4\cdot8}$ in BPA and $10^{2\cdot4}$ in RVF antiserum. No other deaths attributable to RVF occurred.
† Two mice of 5 died. Protection Test Challenge = 80 mouse LD_{50}
†† Three mice of 5 died.

inoculum and the fate of the rats concerned. From this table it may be seen that, with the exception of 2 animals (which received $10^{7\cdot6}$ and $10^{4\cdot6}$ mouse-LD_{50} respectively and whose sera gave inconclusive results) all those animals which received doses of $10^{2\cdot6}$ mouse-LD_{50} or greater had developed neutralizing antibodies for RVF. Of the animals which received $10^{1\cdot6}$ mouse-LD_{50}, one gave a positive result, one gave an inconclusive one, and 3 were negative, while none of those which received $10^{0\cdot6}$ mouse-LD_{50} produced any antibody detectable by our protection-test method.

The results of the first two experiments showed that *Arvicanthis* will circulate RVF virus and a third experiment was designed to show at what time after inoculation, and in what degree, RVF can be recovered. Sixteen known non-immune *Arvicanthis* were taken and were inoculated with decimal dilutions of virus, the dosage levels ranging between 200 and 20,000 mouse-LD_{50}. Thus there were 4 groups each of 4 *Arvicanthis*, these were identified with the letters A, B, C and D. These groups were again subdivided into pairs A_1, A_2, B_1, B_2, etc.* for bleeding purposes. At intervals of 24 hours all sixteen animals were bled, the bloods pooled in pairs as above, and titrated in mice. This procedure was adopted for 5 days; thus, at evenly spaced intervals from 24 to 120 hours after inoculation, 8 titrations were carried out. The results of the titrations are given in Table III together with information relating to the dosage employed for the inoculation of each group.

DISCUSSION

The results given in Tables I and II indicate that *Arvicanthis* which receive an inoculum containing less than 10^1 mouse-LD^{50} of RVF are apparently unaffected, while those which receive $10^{1\cdot5}$ mouse-LD_{50} or more, will almost certainly produce neutralizing antibodies to it, thereby indicating that the virus has undergone multiplication in them. During the post-inoculation period the majority of the rats remain healthy, though a few of them show signs of illness and may

TABLE III. RESULTS OF VIRUS CIRCULATION EXPERIMENT

Rat Pair Identification	*Mouse LD_{50} Inoculated*	*Mouse LD_{50} of Virus 1 ml*				
		Day 1	*Day 2*	*Day 3*	*Day 4*	*Day5*
A_1	$10^{3\cdot3}$	$10^{2\cdot5}$	*	*	*	*
A_2	$10^{3\cdot3}$	*	*	*	*	*
B_1	$10^{4\cdot3}$	*	*	*	*	*
B_2	$10^{4\cdot3}$	*	*	$10^{3\cdot7}$	$10^{2\cdot5}$	$10^{2\cdot1}$
C_1	$10^{3\cdot3}$	*	*	*	*	*
C_2	$10^{3\cdot3}$	$10^{2\cdot0}$	*	$10^{2\cdot0}$	$10^{2\cdot8}$	*
D_1	$10^{2\cdot3}$	*	$10^{1\cdot0}$	$10^{3\cdot0}$	$10^{2\cdot7}$	$10^{4\cdot5}$
D_2	$10^{2\cdot3}$	*	$10^{1\cdot0}$	$10^{4\cdot0}$	$10^{3\cdot1}$	$10^{2\cdot8}$

* = No virus detected

die. Those which die circulate virus in their peripheral blood at levels as high as $10^{8\cdot0}$ mouse-LD_{50} per 1·0 ml. Table III shows that *Arvicanthis* inoculated with varying doses of RVF may circulate virus over a period of 5 days following the inoculation. It may also be seen that an inoculum containing 200 mouse-LD_{50} is likely to produce higher and more consistent levels of circulating virus than a larger one. That this is not invariably true is shown by the fact that one animal which received 10^3 mouse-LD_{50} (see Table I) circulated $10^{9\cdot0}$ LD_{50} per ml. of blood, and another (see Table II) which received $10^{7\cdot6}$ mouse-LD_{50} circulated $10^{6\cdot3}$ mouse-LD_{50} per ml. of blood. Table III also shows that the highest level of circulating virus is attained on the 3rd post-inoculation day and that this level is likely to reach, if not to exceed, $10^{5\cdot0}$ mouse-LD_{50} per ml. of blood in *Arvicanthis* which remain perfectly well during the period of viraemia.

Using the figures given above, and the fact that an average-sized mosquito in this area usually takes a blood meal of about 4 mg.,[5] we shall now attempt to show that the *Arvicanthis* population living in this area are capable of taking part in, and, in fact, maintaining a mosquito-mammal cycle for RVF.

It was shown that an inoculum containing $10^{1\cdot6}$ or 40 mouse-LD_{50} is adequate to produce an infection in *Arvicanthis*. If no allowance is made for any virus multiplication in the mosquito, in order to take up sufficient virus to constitute one infecting feed, the insect would have to imbibe blood

* The pairing of the animals and pooling of their blood was resorted to in order that the continuity of the series should not be broken in the event of the death of a rat, which was possible either as a result of the inoculation or from trauma sustained during the daily bleeding.

containing not less than $40 \div 0{\cdot}004$ or 10^4 LD_{50} per ml. It has also been shown that infected, though apparently well, *Arvicanthis* are capable of circulating RVF virus at levels in excess of 10^5 mouse-LD_{50} per ml. of blood. The *Arvicanthis* are therefore able to supply the minimum infecting mosquito feed as outlined above. It is, however, exceedingly unlikely that any mosquito which takes part in a cyclical transmission series, will not contribute to the cycle by allowing the multiplication of the virus within it, so that in all probability a circulating titre rather less than 10^4 LD_{50} per ml. will suffice to infect a mosquito.

Ross and Gillett[6] showed that a circulating virus titre of $10^{2{\cdot}3}$ in the blood of a grivet monkey *Cercopithecus aethiops centralis* Neumann was adequate for the transmission of yellow fever by the mosquito *Aëdes* (*Stegomyia*) *africanus* Theobald. If we may draw an analogy between this cycle and a hypothetical one in which there is cyclical transmission of RVF virus through *Arvicanthis* and a mosquito, it would appear that the titres of circulating virus found in the *Arvicanthis* which survived inoculation are adequate to maintain the cycle.

There is little doubt that the titre of virus in the blood of those *Arvicanthis* which die after an infection with RVF is quite adequate for the infection of a mosquito which bites them when moribund or newly dead. This state of affairs, however, does not fill the requirements of a natural host for, if a species is highly susceptible to an agent, the population is decimated in the epizootic which follows its introduction, and any survivors are likely to have become immune to it. If, however, a species which has a high population turn-over resulting in a continuous supply of non-immune animals which usually remain relatively unaffected by an infection with RVF virus, though they circulate it in a concentration great enough to infect a haematophagous arthropod vector, then the species is able to act as a natural host. The only other essential requirement is that the species concerned has a sufficiently wide distribution.

It is the purpose of this communication to put forward the suggestion that the field rat *A.a.nubilans* fulfils all the requirements necessary for it to be a natural host of RVF in Abyssinia, Kenya, Uganda, Tanganyika and the Belgian Congo, in all of which territories it is found in abundance. It is also felt that the closely related species, *Arvicanthis niloticus* Desmarest, which occurs in Egypt, Portuguese Guinea, the Sudan, Nigeria, Sierra Leone, Tanganyika, Uganda, the Gold Coast and French West Africa, and *Arvicanthis tenebrosus* Kershaw which occurs in Tanganyika and Northern Rhodesia, might well behave in a similar fashion to *A.a.nubilans* and that any or all of these species might take part in the dissemination of RVF while acting as a natural host.

Further work is planned to investigate the ability of various other rodent species to behave in this manner, with particular reference to the genera *Lemniscomys* and *Mastomys* which are known to extend as far south as the Cape Province in the Union of South Africa.

SUMMARY

A series of experiments which show that the field rat *Arvicanthis abyssinicus* is able to circulate Rift Valley Fever virus, without itself succumbing to the infection, have been described. An attempt has been made to show that *A. abyssinicus* is likely to play a part in the cyclical transmission of RVF in which a haematophagous arthropod is also involved. It has also been suggested that 2 closely related species *A.niloticus* and *A.tenebrosus* may well behave similarly. As there is considerable overlap in the territories in which these species are found, it has further been suggested that they might play a part in the dissemination of RVF virus.

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DERMATOMYOSITIS

CASE REPORT

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This case is reported firstly because it shows several transitional features between dermatomyositis and disseminated lupus erythematosus, and secondly because of the occurrence of heart failure and its dramatic response to cortisone therapy.

It is not intended to discuss in any detail the clinical or pathological features of dermatomyositis, as these are well documented in the literature. Keil (1940 *a* and *b*) discusses fully the differentiating features between dermatomyositis and disseminated lupus erythematosus and points out that in many cases—the transitional cases—it is difficult to decide which of the two diseases one is dealing with. The clinical and pathological features of this case, however, favour the diagnosis of dermatomyositis.

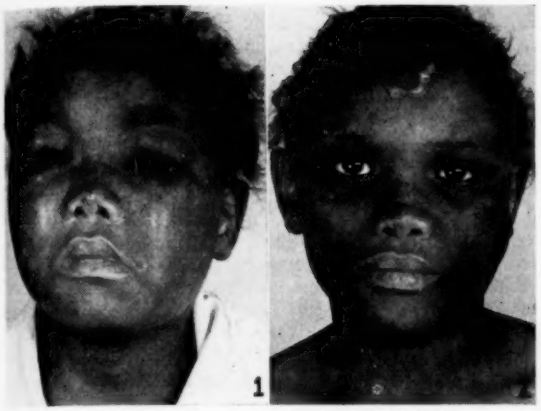

Fig. 1. Facial appearance on admission.

Fig. 2. Facial appearance, showing disappearance of oedema, healing of skin lesions and wasting of facial muscles, 2 weeks after commencement of cortisone therapy.

CASE REPORT

A female Eurasian child of 9 years was seen by us for the first time when she was admitted to Baragwanath Hospital on 6 August 1953. The mother stated that her child had been quite well until January 1949, when she noticed that the lower eyelids had become darkly pigmented. This persisted, and in January 1950 a small dark patch appeared on the side of her nose. A few months later this subsided but did not disappear altogether. Similar patches then appeared on her cheeks, trunk and arms. They were not itchy or painful and were not aggravated by sunlight. At the same time she developed swelling of her face, trunk and legs. The swelling did not involve all the parts of her body simultaneously nor, having developed, was it permanent. For example, the swelling of the legs would subside for a few weeks while that of the face would get worse. She was seldom entirely free from swelling at one time. On one occasion the oedema of the vulva and perineum made micturition and defaecation difficult. The child had become increasingly listless and easily tired and her appetite was poor. The hair of her head had been falling out for 8 months before admission. At no time was there any difficulty in swallowing. The child had never complained of sore throats, joint pains, or breathlessness.

On Examination

The outstanding feature was the gross oedema of the face (Fig. 1). The eyelids were swollen and purple in colour (the so-called heliotrope pigmentation, as described in dermatomyositis). The palpebral fissures appeared as slits and she could not open her eyes. The scalp hair was thin and dry and there were large areas of alopecia. The skin was generally dry and there were patches of desquamation mainly involving the arms. There was a rash over the nose and cheeks, in the so-called 'butterfly' distribution, consisting of patches of pigmentation with induration of the underlying tissues of the face. Patchy pigmentation occurred on the arms and the trunk, varying from a few millimetres to approximately 5 cm. in diameter. The underlying tissues were indurated but not tender. There was brawny oedema of the arms and thighs, with some pitting oedema around the ankles. Slight peri-ungual pigmentation was noted. A few slightly enlarged non-tender glands were present in the neck and in both axillae.

The heart was not enlarged but a tachycardia of 120 beats per minute, and a triple rhythm were present. The temperature was 99°F, and the blood pressure 110/70 mm. Hg.

The liver edge was felt 2 finger-breadths below the costal margin. The spleen was not palpable. There was generalized weakness of her arms and legs, but the deep reflexes were brisk and muscle wasting was not detectable, probably owing to the overlying oedema.

Progress

A low-grade pyrexia persisted during most of the child's 3 months' stay in hospital, the temperature varying from 97° to 101°F, with occasional spikes to 104°.

An essentially clinical diagnosis of dermatomyositis was confirmed by a skin and muscle biopsy reported by Dr. Pepler of the South African Institute for Medical Research as showing histological features characteristic of that disease. These features were well described by O'Leary (1949)—see below.

Therapy was begun on 8 August 1953 with ATCH, 20 units 6 hourly, by intramuscular injection. A mild improvement was evident after 3 days; the oedema subsided but the pyrexia and tachycardia persisted. Two weeks after admission, on 21 August 1953, the child became breathless at rest and began coughing blood-stained sputum. By the next day there was evidence of severe congestive cardiac failure and the patient appeared moribund. The pulse rate was 180 a minute. A rise of venous pressure was noted. The heart was now detectably enlarged and the blood pressure was 120/100 mm. Hg. The liver was enlarged to 4 finger-

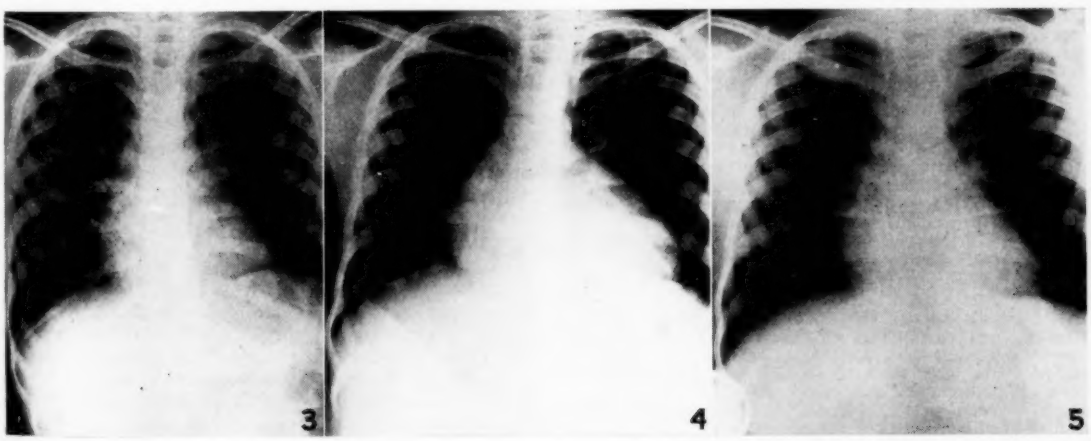

Fig. 3. X-ray of chest showing heart size on admission.

Fig. 4. X-ray of chest showing heart size 1 week after commencing cortisone therapy.

Fig. 5. X-ray of chest showing great reduction in heart size while maintained on cortisone therapy.

breadths below the costal margin, and was very tender. Crepitations were present at both lung bases. On auscultation of the heart a gallop rhythm was heard, but no cardiac murmurs. It was felt that ACTH therapy may have been a factor in the production of cardiac failure and it was therefore discontinued. However, no improvement occurred on routine therapy with digitalis and mercurial diuretics. In the absence of hypertension or any electrolyte disturbance, cortisone therapy was begun on 24 August with a dose of 100 mg. followed by 25 mg. every 6 hours. A dramatic improvement occurred. Within 24 hours there was a rapid disappearance of the oedema, reduction of the venous pressure, and slowing of the heart. The cardiomegaly, however, persisted for about 2 weeks. During this episode serial electrocardiograms showed a low voltage and generalized inversion of T waves. Radiography demonstrated generalized enlargement of the heart which, with the subsequent return to normal, is well shown in Figs. 3, 4 and 5.

On continued cortisone therapy, the child's condition steadily improved. The disappearance of oedema revealed severe muscular atrophy, especially marked in the face and arms, where pits were left in the subcutaneous and muscular tissues under pigmented skin patches (Fig. 2). On 7 November 1953 the patient was discharged from hospital. She was apyrexial and the alopecia had disappeared. She was maintained on a dose of 25 mg. of cortisone daily and was seen at the out-patient department at weekly intervals.

Other Investigations during the First Admission

Blood. Haemoglobin 10·8 g.%. Total white-cell count 7,400 per c.mm. (neutrophils 68%, monocytes 6%, lymphocytes 25%, eosinophils 1%). MCHC 33%. Blood urea 37 mg.%. Wassermann reaction negative. Serum albumin 1·7 g.%, serum globulin 4·4g.%. Blood cholesterol 130 mg.%. L.E. cells were sought for in the peripheral blood on several occasions but were not found.

The *urine* was of normal specific gravity and on occasion contained a trace of albumin. Microscopy revealed 4 red blood-cells and 5 white blood-cells per high-power field.

Second Admission

The patient was readmitted on 26 December 1953, complaining of a rash at the tips of her fingers and a recurrence of alopecia, fever and facial swelling. She had noticed bluish marks at the tips of her fingers, which had developed into hard, painful blisters. On examination her face was as swollen as on the first admission. There were small necrotic reddish-brown indurated areas on the tips of all her fingers and just proximal to these were small bluish blisters. The blood pressure was 110/70 mm. Hg. and the heart was normal in size. This relapse was probably precipitated by a failure to attend regularly for maintenance therapy.

Treatment was started with 50 mg. of cortisone 8 hourly and 'Corticotrophin Z', 20 mg. on alternate days. This time, however, there was no clinical response. Her right arm became grossly swollen and numerous bullae developed on the face, forearms and elbows. These ulcerated and became necrotic. A persistent pyrexia of 100-104°F continued until her death on 20 January 1954, 24 days after admission. A rapid increase in dosage of cortisone up to 500 mg. daily, in addition to 400 mg. of hydrocortisone daily, failed to prevent the fatal outcome.

Additional Investigations. The total creatinine in a 24-hour specimen of urine was 342 mg. The pre-formed creatinine amounted to 293 mg., giving a daily creatine output of 49 mg. On 7 January L.E. cells were detected in the peripheral blood by Dr. R. Cassel of the South African Institute for Medical Research.

Necropsy

Apart from the external skin changes described above, there were no macroscopic findings of significance. The following *histological report* was submitted by Dr. Pepler:

'Section of the lungs shows congestion with extravasation of red cells and oedema fluid into the alveoli. In addition there is an acute bronchiolitis The kidney is congested and shows occasional albuminous casts in the collecting tubules. The liver is moderately congested and there is considerable fatty change involving the majority of the parenchymal cells. No significant lesion has been observed in the heart apart from one small focus of mycocardial haemorrhage. No lesion has been observed in the suprarenal glands. A section from the elbow region of skin and muscle shows oedema and necrosis of the subcutaneous tissue with diffuse focal infiltration by lymphocytes, plasma cells and histiocytes and polymorphs. The small arteries show hyperplasia of the intima and fibrinoid change. The underlying muscle shows degeneration and necrosis with infiltration by chronic inflammatory cells. A section of skin, subcutaneous tissue and muscle from the leg showed similar changes but with much more marked muscle degeneration and necrosis. The features are those of dermatomyositis.'

DISCUSSION

The diagnosis in this case is based on the extremely characteristic facial appearance, the alopecia, the gross peripheral oedema and the demonstration later of marked muscular atrophy. Sheard (1951) states that the rash in lupus erythematosus may spread from the 'butterfly area' of the face to involve the eyelids, but does not involve the eyelids first. It may very rarely be associated with facial oedema as in the related condition known as erysipelas perstans faciei (Gold and Gowing 1953).

A feature of the case which suggested disseminated lupus erythematosus was the periungual discoloration and later the atrophic rash with blisters at the fingertips. However, Keil (1940 *b*) has described atrophic areas over the small articulations of the fingers and 'peri-ungual changes' in dermatomyositis.

Lupus erythematosus only rarely affects large muscle-masses, causing the severe wasting seen in this case.

With regard to the histological findings, there is some difference of opinion about the interpretation of muscle changes in dermatomyositis. O'Leary (1949) describes the pathology as follows: 'The first changes appear to occur within the bundles of the muscles themselves, with proliferation of the nuclei, effacement of the transverse striations, separation of the myofibrils and occasionally coagulation and hyalinization of the sarcoplasm. Later there may be fragmentation of the fibrils, granular, fibrinous and vacuolar degeneration.' On the other hand, Jager and Grossman (1944) and Pagel *et al.* (1949) state that similar histological changes occur in dermatomyositis, lupus erythematosus, acute rheumatic fever, scleroderma and other conditions. In lupus erythematosus it is generally accepted that vascular changes predominate, i.e. fibrinoid necrosis of the blood vessels, and that although there may be microscopic evidence of muscle damage, there is rarely clinical evidence of muscle atrophy.

In view of the heart failure, we were surprised that no significant histological lesion could be demonstrated in the myocardium. Although myocardial involvement occurs fairly often in dermatomyositis, heart failure is rare, and we could find no report of heart failure that had been successfully treated. In 40 cases reported by O'Leary and Waisman (1940), 19 deaths took place; amongst these, one patient died of cardiac failure, in 9 cases there was clinical and electrocardiographic evidence of cardiac abnormality, and cardiomegaly was reported in 5 cases and myocarditis in 2 cases. Keil (1940 *b*) states that myocarditis is more frequent than is commonly believed. 'Persistent tachycardia is often observed even in afebrile cases, and in other cases the patients may, have dyspnoea, oedema, cyanosis and arrythmias.'

There are two possible explanations for the appearance of L.E. cells in this case. Either this is a transitional case similar to those described by Keil, or the finding of L.E. cells was related to the cortisone therapy. Slocumb (1953)

reported the demonstration of L.E. cells in 15 patients with rheumatoid arthritis on the reduction cortisone dosage. In many cases of lupus erythematosis, L.E. cells are found only after ACTH or cortisone therapy and it is possible that the hormones may stimulate the production of the plasma factor essential in the production of these cells. L.E. cells have also been found in other conditions, such as the leukaemias, multiple myelomatosis (Beerman—1951), and after penicillin therapy.

The finding in this case of a normochromic anaemia, low white-cell count and a mild monocytosis corresponds with the findings in O'Leary's cases. There was a reversal of albumin/globulin ratio, although the globulin level was not unduly high. This derangement of the serum proteins is difficult to interpret; it is a common finding in this hospital.

SUMMARY

A case diagnosed as dermatomyositis is described. The outstanding features were:

1. The occurence of cardiac failure with gross cardiomegaly, both of which responded to cortisone therapy.

2. The occurence of L.E. cells in the peripheral blood.

Some of the distinguishing features between dermatomyositis and disseminated lupus erythematosus are discussed.

We are indebted to Dr. L. Hirsowitz, under whose guidance the patient was treated, and to the Medical Superintendent, for permission to publish this case; to Dr. S. Wayburne for the clinical photographs, and to Mr. A. Shevitz for reproductions of the X-rays.

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UMBILICAL HERNIA IN THE BANTU

A CASE OF STRANGULATION

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J. H. YOUNGLESON, M.B., CH.B. (CAPE TOWN)
Kokstad

The stimulus to the present investigation was the occurrence of a strangulated umbilical hernia in a 4-year-old Bantu male infant. The strangulation was of the Richter's type, the hernia orifice admitting 2 fingers. Struck with the rarity of strangulation in these hernias, we decided to investigate the incidence of umbilical hernia in the Bantu.

Methods and Materials

A series of 1,030 consecutive African patients was examined by ourselves and our partners working in general practice in East Griqualand and Pondoland. Every African presenting himself for any complaint whatever was examined for umbilical hernia; the figures were augmented by 70 adult males and 24 females from the local gaol.

Classification

We divided the umbilical hernias into 3 grades:

Grade I: Cough impulse only (more than one cough may be required to distend the sac). In such hernias the orifice is too small to admit anything more than peritoneal fluid, so that this grade cannot lead to complications.

Grade II. Admits the tip of a finger. Such hernias may or may not contain abdominal contents.

Grade III: Larger than the tip of a finger. Such hernias always contain abdominal contents. They occasionally admit up to 3 or 4 fingers.

All parous women with hernias gave a history of the lump having been constantly present since childhood.

Results

The incidence of umbilical hernia of the 3 grades in our 1,030 patients classified by age and sex, is shown in Table I and Fig. 1. A graph in Fig. 2 shows the incidence of umbilical hernia (all grades) according to age; corresponding graphs

TABLE A. RESULTS

	0–1		1–3		3–5		5–7		7–9		9–16		16+	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Total Cases seen	133	114	75	40	29	32	25	16	6	14	16	33	172	325
Hernia Grade I No.	42	32	25	12	11	9	8	3	1	2	2	4	13	34
%	31	28	33	30	37	28	32	19	17	14	12	12	8	10·4
Hernia Grade II No.	29	27	14	7	5	4	1	2	—	1	—	3	5	11
%	22	23	19	17	38	13	4	12	—	7	—	10	3	3·4
Hernia Grade III No.	16	18	2	1	—	2	1	—	—	—	1	2	2	6
%	12	16	3	3	—	6	4	—	—	—	6	6	1	2
Total Hernias No.	87	77	41	20	16	15	10	5	1	3	3	9	20	51
%	66	68	55	50	55	47	40	31	17	21	18	28	12	16

Total cases seen, 1,030. Total hernias, 358.

are also shown reflecting published figures for other population groups (see below).

Conclusions

1. We found a relatively high incidence of umbilical hernia (67%) in children under 1 year, falling gradually with increasing age to leave a surprisingly high residual incidence of hernias (mostly grade I) in adults. Probably the grade-I and grade-II hernias tend to close spontaneously, while the gross grade-III hernias tend to become smaller but persist into adult life. In our small series the incidence of grade-III hernias under 1 year exactly equals the over-all incidence of hernias in adults.

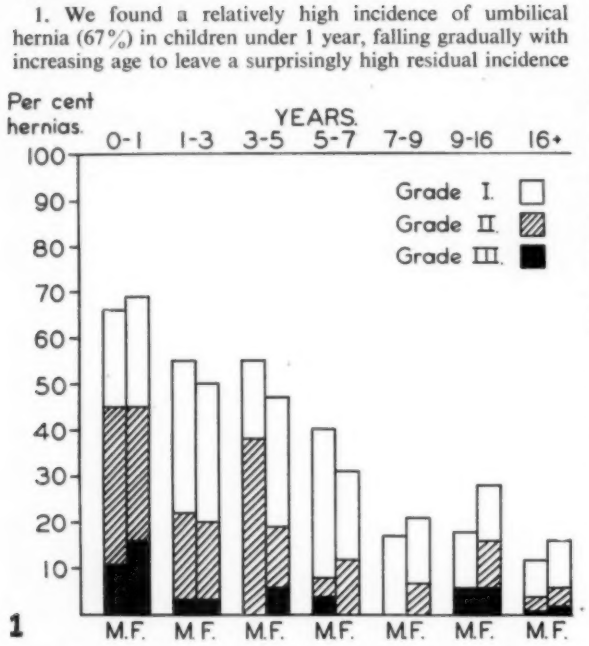

Fig. 1. Umbilical hernia. Graphic analysis of percentages, by sex, age and grade.

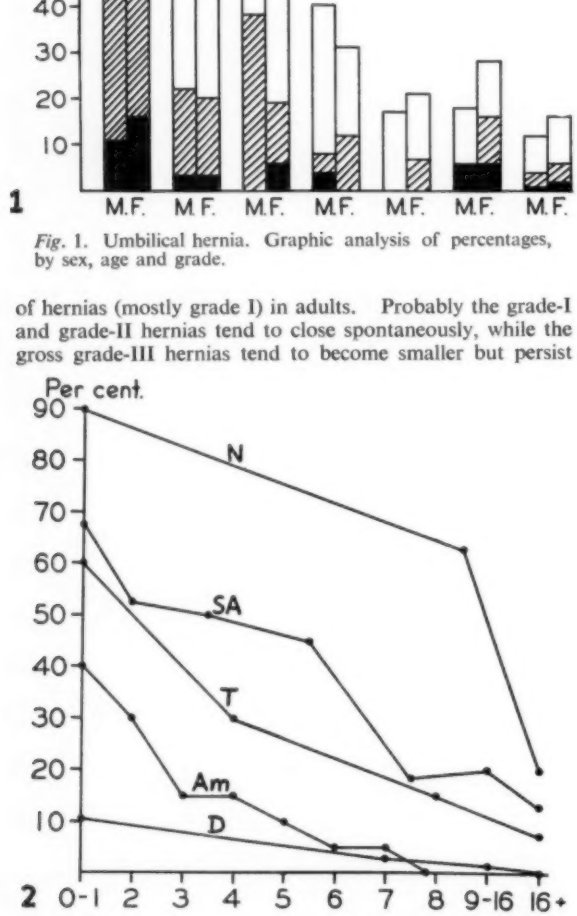

Fig. 2. Umbilical hernia. Charts of percentages, by age. N=Africans in Nigeria. SA=Africans in South Africa. T=Africans in Tanganyika. Am=American Negroes. D=Europeans in Denmark.

2. As regards the sex incidence, in infants and children under 9 years we find the umbilical hernia to be slightly commoner in males, whereas in older children and adults it is the females who show the higher incidence. It will be noted that the excess in females applies in the 9-16 age group as well as in the child-bearing ages. All parous women with umbilical hernia gave a history of the lump having been constantly present since childhood. As far as could be ascertained, no hernias in adult females were due to child-bearing.

3. During the past 3½ years, we have seen about 13,000 Bantu with umbilical hernias. Only one of these has strangulated.

DISCUSSION

The results from other parts of the world are as follows:

Riisfield[1] found the following incidence of umbilical hernia in Danish children:

1 year	11·5%
7 years	4%
14 years	0·25%

Mack[2] in a series of Africans in Tanganyika found:

0– 1 year	60%
4 years	30%
8–10 years	20%
15–20 years	7%
20+ years	6%

E. Perry Crump[3] in American Negro children found:

0–1 year	40%
1– 2 years	30%
3 years	15%
4 years	15%
5 years	10%
6 years	5%
7 years	5%
8–16 years	0%

(In this series a hernia was considered present only if it admitted the tip of a finger, or if it permitted the extension of intra-abdominal contents; i.e. it excluded our grade-I hernia).

Jelliffe[4] in a series of Nigerian Natives found:

0– 5 years	91%
6–15 years	64%
Adults (educated)	14%
Adults (uneducated)	27%

Fig. 2, giving a graphical comparison of these figures with our own, shows that:

1. All series give a far higher incidence in the African than in the European.

2. There is a fairly wide variation in the figures obtained from various parts of Africa and America. We feel that differences of interpretation between individual observers and their varying definitions of a minimal hernia may be a confusing factor in comparing these figures. Our percentage incidence in the 5-7 and 7-9 year groups is based on somewhat small numbers.

The outstanding facts arising from this investigation are that in the African, umbilical hernias are very common in

infancy; the bulk of them close spontaneously and strangulation is extremely rare. For these reasons, we feel justified in disagreeing with those text-books that advise repair in all children over the age of 3 years with an umbilical hernia admitting the tip of a finger. In the Bantu, surgical repair of uncomplicated umbilical hernia is clearly unjustified, and this should apply to Europeans too with the exception of a repair for cosmetic reasons only, in a schoolgoing child.

SUMMARY

1,030 consecutive African patients from East Griqualand and Pondoland were examined for infantile umbilical hernia.

The incidence was found to be 67% in infants under one year, falling to 14% in adults.

This is in contrast to figures for Europeans, where the incidence of infantile umbilical hernia is low.

A case of strangulation is reported, and the great rarity of this occurrence in a community where umbilical hernia is so very common is stressed.

We wish to express our thanks to our partners Drs. W. A. Muir and G. D. Hartley for their assistance in assembling our data, and to Prof. J. H. Louw of Cape Town for helpful criticism of this paper.

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THE PROFESSIONAL PROVIDENT SOCIETY OF SOUTH AFRICA*

W. A. M. MILLER, M.B., B.CHIR. (CANTAB.)

The professional Associations sponsoring Professional Provident Society of South Africa are the Dental and Medical Associations the Pharmaceutical Society, the Societies of Advocates, the Association of Law Societies and the Veterinary Medical Association. This Society is your Society, operating entirely for the benefit of the members of your respective professions, but it is only too apparent that the majority in the professions is unaware of the great advantages membership offers. These advantages are so tremendous that we feel that they should be known to all and we hope that you will assist by stimulating the interest of your colleagues.

We are all professional men and have at least one other thing in common—if we don't work we don't earn. The very purpose of this Society is to reverse this situation, at least if our reason for not working is illness or ill health. The Society is an entirely mutual organization, every member sharing in its profits. It provides for sick-pay cover up to £135 per month at a nominal cost and in addition provides disability benefits and a handsome lump sum payment on retirement.

The present assets of the Society amount to over £200,000. The main reason for the formation of the Society is the fact that examination of sickness insurance policies offered by insurance companies reveals that they afford inadequate cover for those professional people who are entirely dependent on their own efforts for income. These insurance policies are never more than year-to-year contracts, and should the policy-holder's health deteriorate to a degree when he is no longer considered a good risk, he will find that the insurance company is no longer prepared to continue the insurance. In effect, this means that one can only be insured for sickness whilst enjoying the best of health or until one's first serious illness. This is obviously quite inadequate, and no proper safeguard. What is required is continuous insurance, which facility can only be obtained from this Society *where, once a person is admitted to membership, he remains a member until retirement, regardless of the degree to which his health may deteriorate after admission.* There are many persons in the professions who have paid sickness insurance for many years in the belief that this facility would be continued indefinitely. Many have received a rude awakening when told that such insurance is no longer available to them.

Those who are members of the Professional Provident Society have then realized the tremendous difference between the yearly policy and our continuous insurance, and have been very thankful that they still retain the necessary security for themselves and their dependants. In essence the Society guarantees that, once accepted, the member is fully entitled to enjoy the benefits of membership until retirement.

I feel that I must also emphasize that members should be more conscious of the fact that applications for sick benefits are dealt with by their colleagues and friends, who are ever mindful that payment of these benefits is one of the main objects of the Society. I hope that all of you who are eligible for membership will give serious thought to joining the Society. It is certainly in your interests to do so. If you carefully examine the position, I am sure you cannot help coming to the conclusion that it is an organization to which it is essential all professional persons should belong.

Here I would add that it is our duty as senior members of our professions to ensure that the younger members take early advantage of membership and do not leave the question of joining until the time when bad health may prejudice their acceptance.

The more members we have the more evenly will the risks be spread, and the better should be the results. Moreover, the larger the Society, the better will be its position to provide for other benefits. Already we are in a position in some instances to arrange loans to members for home building, at interest rates somewhat lower than the ruling rates, and I should like to add very briefly that we are at present endeavouring to establish a pension fund for self-employed professional persons. We have approached the Government and asked for legislation to exempt from taxation contributions to the proposed pension fund.

* An address delivered to members of the associated professions, Johannesburg, 27 March 1957.

REPORT ON WMA ASSEMBLY, HAVANA†

EMILIA KRAUSE, M.R.C.S., L.R.C.P.

Bloemfontein

I wish to express my appreciation and thanks to your Council for the honour and privelege of having been appointed your delegate to the World Medical Association, which met in Havana in October 1956. This is the 5th occasion in the 9 years' existence of the WMA on which I have been present at meetings in various countries. At first there was much talk on Social Security almost *ad nauseum*. Today the attitude has changed; the doctors of the world have woken up and the medical profession has gained authority by the formation of the WMA. As a profession we are

† Submitted to Federal Council at the meeting held on 27-29 March 1957.

united and strong—Governments are showing greater respect for the WMA and the differences we had with the World Health Organization have been largely overcome. The appointment of a liaison officer, Dr. Jean Maystre, in Geneva, who now is in close contact with the offices of the WHO and ILO helped to bring about this change.

Dr. T. C. Routley, of Canada, will be attending our Medical Congress at Durban this year and will speak to the Medical Association of South Africa on what the WMA means. I feel that South Africa should support the WMA in every way and strengthen the bond of international relations in the medical profession. We are a unique brotherhood; without our services nations, governments and politicians would crumble. Let us stand together and not be swayed by a materialistic world. The medical profession stands for World Peace.

I was invited to attend the Secretariat Liaison Conference held at the WMA Secretariat in New York on 19-20 October 1956. We were shown the offices and the work that is done in the different departments. It is a complex organization run most efficiently with Dr. Louis Bauer in charge. The language question—French, Spanish, English—complicates the cost of the Journal and of conferences. Expert translators must be employed.

Ways and means are sought to reduce the costs but, with the demands of 53 member associations, we can no longer expect the United States Supporting Committee to provide the majority of finances to carry on the activities of the WMA. Each country will have to contribute a greater share in future.

Dr. Bauer gave an excellent summary of the aims and achievements of the WMA since its organization in 1947. For those in South Africa who may not know, the WMA was organized by the national medical associations with the following aims:

1. To protect and promote the freedoms essential to effective medical practice.

2. To raise standards of medical education, medical care and health throughout the world.

3. To bring together the doctors of the world to share knowledge and ideas and to discuss and provide the solution of problems common to medicine the world over.

4. To speak for the doctors of the world before other international organizations concerned with health and medical care.

This Conference served a very useful purpose in that there was an intimate exchange of ideas and suggestions. Mr. James E. Bryan, not himself a doctor, gave a paper on 'Basic Principles of Medical Public Relations' and a number of other interesting papers were presented.

These are but a few personal impressions gained at this most interesting Assembly of the World Medical Association.

IN MEMORIAM

HENRY E. SIGERIST

Dr. A. Jokl of Johannesburg writes: On 17 March 1957 one of the greatest scholars of our time, Professor Henry E. Sigerist, died in Pura, a small village in Switzerland.

He was born in Paris on April 7 1891 of Swiss parents. After the early death of his father, his widowed mother moved with her children back to Switzerland (1901). He went to the gymnasium in Zurich and later studied medicine at the University there. At the same time, he familiarized himself with old and oriental languages. For some time, he worked at the Universities of Munich and of London. In 1917 he qualified in medicine and in 1921 became lecturer at the Zurich University.

Dr. H. E. Sigerist

His great gifts as a teacher, and his researches, soon attracted the interest of the scientific world. In 1925 he was called to Leipzig as successor of Karl Sudhoff, at that time the leading medico-historian of Europe. In 1932 he moved from Leipzig to Baltimore, to become the leader of the large historical institute at Johns Hopkins University, where he worked for 15 years. During this period he travelled extensively, visiting, amongst other countries, Yugoslavia, Canada, India, Soviet Russia and South Africa. While in South Africa he gave a series of lectures and was awarded an honorary doctorate by the University of the Witwatersrand (1939).

Sigerist was a prolific writer. His best known works are: *Great Doctors, Introduction to Medicine, Civilization and Disease* and *Socialized Medicine in the Soviet Union.* Smaller papers published by him must number several hundreds. Many of them are found in the series *Monumenta Medica*, founded by him, and in the *Bulletin of the Johns Hopkins Institute of the History of Medicine.*

In 1947 he decided to relinquish his academic post and to retire. He had planned to write a comprehensive History of Medicine in eight volumes. The Yale University considered this project important enough to be subsidized, and appointed him Research Associate with a salary and the freedom to work where he liked, thus enabling him to devote all his time to this task. In 1951, the first volume of this monumental work was published. It was greatly admired by all who take an interest in this branch of science. The next volumes were eagerly awaited, but alas, no more were to come. In 1954 he became seriously ill and though, after a few months he recovered somewhat—his career as a scientist was ended.

The importance of Sigerist lies in the encyclopaedic knowledge he brought to his writings. Apart from being a doctor of medicine and well acquainted with medical progress during his lifetime, he was a classical scholar, a linguist with a working knowledge of at least 14 languages, and a student of philosophy, general history, archaeology, geography, anthropology and art. In addition, he was greatly interested in the relationship of medicine to the social problems of our time. His approach to Medical History was therefore on a much broader basis than had ever been attempted before.

The universality of his mind enabled him to see the philosophical background of historical events and to assess and to interpret them according to their value, not only for the individual patient, but for the nations and for mankind in general. His work has remained a torso and one fears there is nobody living who can complete it. Sigerist was probably the last polyhistorian in the true sense of the word. He had other outstanding qualities as well. He was an excellent speaker and his lectures everywhere drew large audiences.

Though he was an international figure, he was simple, cheerful, approachable and helpful. Busy as he was, he had always time to see his friends and pupils and to give them encouragement and assistance. Everybody who has known him, who has read some of his books or listened to his lectures, and particularly those who have been in close personal contact with this captivating personality, will mourn his death as an irreparable loss.

JOHN OLOFF MARAIS, M.B., CH.B. (EDIN.)

Prof. F. O. Fehrsen, of Cape Town, writes: Dr. John Oloff Marais, who had only recently retired from general practice at Middelburg (Cape), met his death under very sudden and tragic circumstances. He was knocked down by a motor car in Stellenbosch on 4 April, and died on the following day without regaining consciousness.

Dr. J. O. Marais

A son of the late Prof. J. I. Marais of revered memory, he was a graduate of the Stellenbosch University. He qualified M.B., Ch.B. of Edinburgh in 1916. After the usual hospital appointments he settled in Middelburg, where he carried on a large and successful practice for nearly 40 years. He was possessed of a happy and very attractive personality and soon endeared himself to his patients and indeed to all who were privileged to know him. He symbolized the family doctor in the finest tradition. He always placed service before self and, to him, material success was of minor importance. He will be affectionately remembered by his patients, colleagues and friends.

For the greater part of his life Dr. J. Marais was dogged by ill-health, but he faced this disability with characteristic cheerful fortitude and continued to serve his patients with a selfless devotion and a complete disregard of himself. His courage in these adverse circumstances had its origin in an unobtrusive but nevertheless deeply rooted religious faith. He was a man of wide cultural tastes and was particularly fond of music. In the medical sphere he was well read and took a pride in trying to keep abreast of recent clinical developments and scientific progress.

John Marais had the great misfortune and sorrow to lose his wife about 18 months ago. To his two sons who survive him and to his brother, Dr. D. P. Marais, and his sisters we offer our very sincere sympathy in their loss.

In the presence of a large and representative gathering he was laid to rest next to his parents in the cemetery of his old University town.

Dr. I. H. Mathieson, of Middelburg, Cape, writes: For over 37 years, Dr. John Marais served all sections of the community in Middelburg as 'house doctor' in the very broadest sense of the term. His modesty, natural charm and sympathetic approach endeared him to all his patients.

A devoted family man of wide culture, he found time for several interests outside his home. The chief of these was probably Freemasonry. He was twice Master of Unity Lodge, Middelburg, and latterly Officer of District Grand Lodge, Eastern Province. He was also President of the Middelburg Bowling Club for a number of years.

It is sad to reflect that he was spared for so short a time after his retirement to enjoy the leisure that he so richly deserved.

He was a loyal friend with a most delightful sense of humour, and we shall miss his kindly blue eyes and ready smile.

On behalf of the Middelburg Division of the Cape Eastern Branch, I extend deep sympahty to his family in their loss.

ANNOTATION

THE DAY HOSPITAL AND THE NIGHT CENTRE

The 'Day Hospital' which was originated at the Allan Memorial Institute of McGill University and the Royal Victoria Hospital, Montreal, Canada, in 1946, has now been established in various parts of the world, including, not only the US and Britain, but also South Africa. Moross[1] described the 'day-patient' system at the Tara Hospital, Johannesburg, at the South African Medical Congress, Port Elizabeth, in June 1954.

At the Montreal centre the patients come at 9 a.m., receive all forms of treatment which they ordinarily would receive in a day-and-night hospital, and return to their homes at 5 p.m. All types of patients are admitted with the exception of those whose behaviour would be too disturbing to the other patients and those with active suicidal tendencies. The success of the Day Hospital depends primarily upon the following 3 factors: 1. A Day Hospital is very economical to run and reduces the cost of hospitalization to the patient by one-half or two-thirds. 2. The patient remains in close contact with the family throughout the period of treatment and the undesirable effects of dependency upon the hospital are avoided. 3. The Day Hospital is economical in the use of space. Twice as many patients can be cared for in the same floor area as in the day-and-night hospital. If the same floor space is also used for a night centre the economy is still greater.

In 1954 in addition to the Day Hospital a 'Night Treatment Centre' was established in the Montreal General Hospital. The patients are admitted to this every evening at 5.30 p.m., given treatment during the evening hours, remain overnight in the Night Centre, and then return to their work next morning. This type of facility permits the giving of sub-coma insulin, electroshock treatment, psychotherapy, group psychotherapy, narcosynthesis, and the use of various chemical treatments. It is particularly valuable in providing treatment for patients who can at the same time carry on with their jobs. The centre is open 5 nights a week, but not over the week-ends, so as to allow patients to carry on normal social activities. The duration of stay is usually 3 weeks for male patients and 4 weeks for female patients.

1. Moross, H. (1954): S. Afr. Med. J., **28**, 886.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

'Acalo' (Phenaglycodol, Lilly)

This drug has been discovered and developed by Eli Lilly and Company, who supply the following information:

This new, mild, tranquillizing drug calms anxiety states in patients without affecting their mental alertness, sensitivity of perception, or dexterity—all factors of importance in performing such activities as driving cars or operating machines in industry. Its lack of effect on physical and mental skills (with recommended doses) was established in objective and standardized tests giving quantitative results. The tests were performed by Ralph M. Reitan, Ph.D., psychologist at Indiana University, Bloomington, Indiana, USA.

The compound has demonstrated its safety in research with animals and in studies by more than 1,000 clinical investigators. So far, no contra-indications or serious side-effects have been observed with recommended doses.

Chemically, 'Acalo' is 2-p-chlorophenyl-3-methyl-2, 3-butanediol. This is a completely new chemical structure in the

tranquillizer field. It is one of a series of butanediols first synthesized by Lilly's organic chemists. All these compounds had anticonvulsant and hypnotic activity in animals, but 'Acalo' was found to combine highly effective action with a wide margin of safety. The drug appears to act as an interneuronal blocking agent.

In his controlled study, Dr. Reitan gave a series of tests to 14 patients who had been tranquillized with 'Acalo' for a week. The tests were made to evaluate such reactions as response time, time-sense judgment, and rapidity of finger tapping. No untoward effect on the patient's ability to perform these tests was discernible. Measurements of near and distant vision in conjunction with this same study revealed no alteration in visual acuity. The same standardized tests were able to show interference with the established normal abilities of individuals in a group purposely overdosed with 'Acalo'.

'Acalo' is indicated when a pacific effect is desirable. It is beneficial in patients who are afflicted with emotional instability, anxiety-tension states, or functional disorders. Although it usually will alleviate emotional tension, it will not eliminate the abnormalities or basic difficulties that gave rise to the symptom. Consequently 'Acalo' will serve effectively as adjunctive, rather than definitive, therapy. Clinical evidence indicates it will be useful as an adjuvant in patients suffering from a wide range of clinical disorders involving emotional disturbances.

'Acalo' is relatively free from undesirable side-effects. Its low toxicity and its relative freedom from the production of euphoria, tolerance, and physical dependence makes it suitable for treating patients where prolonged use is required.

Each turquoise and white pulvule contains 300 mg. of 'Acalo'. For most patients 1 pulvule 3 times daily will suffice. In certain instances, a patient may take 2 pulvules before retiring to promote a restful night's sleep.

Pulvules 'Acalo' are supplied in bottles of 20.

PASSING EVENTS : IN DIE VERBYGAAN

Woodstock Railway Disaster. The Medical Superintendent of the Woodstock Hospital, Cape, writes: 'On behalf of the staff of the Woodstock Hospital I should like to thank all medical practitioners who offered their services to help deal with the people injured in the railway accident.'

Woodstock-Spoorwegramp. Die Mediese Superintendent van die Woodstock-Hospitaal, Kaap, skryf: ‚Namens die staf van die Woodstock-Hospitaal wil ek graag deur middel van u blad my dank betuig aan alle mediese praktisyns wat hulle dienste aangebied en hulp verleen het in verband met behandeling aan die beseerdes wat in die spoorwegongeluk betrokke was.'

* * *

Dr. W. Grundill, M.B., Ch.B. (Rand), F.R.C.S., F.R.C.S.E., who recently returned after some years of study and practice in thoracic surgery at Harefield Sanatorium in the United Kingdom, now practises at 6 West Burger Street, Bloemfontein. Telephone, rooms 3610. This telephone number is not in the current telephone directory.

* * *

Union Department of Health Bulletin. Report for the 6 days ended 10 April 1957.

Plague, Smallpox, Typhus Fever: Nil.

Epidemic Diseases in Other Countries.

Plague: Nil.

Cholera in: Calcutta, (India); Chalna, Chittagong, (Pakistan).

Smallpox in: Rangoon, (Burma); Ahmedabad, Allahabad, Bombay, Calcutta, Jodhpur, Kanpur, Madras, Nagapattinam, Visakhapatnam, (India); Makassar, (Indonesia); Baghdad, Mosul, (Iraq); Chalna, Dacca, Karachi, (Pakistan); Dar-Es-Salaam, (Tanganyika).

Typhus Fever: Baghdad, (Iraq); Alexandria, (Egypt).

Union of South Africa. Department of Health. Notification of formidable epidemic diseases and poliomyelitis in the Union during the period 11 April to 15 April 1957.

Poliomyelitis

	Eur.	*Nat.*	*Col.*	*Asiat.*	*Total*
Transvaal ..	20	9	1	1	31
Cape Province ..	6	1	6	—	13
Orange Free State ..	1	1	—	—	2
Natal	2	4	—	1	7
Totals	29	15	7	2	53

Plague, Smallpox, Typhus Fever: nil.

* * *

Lede word daaraan herinner dat hulle die Sekretaris van die Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, sowel as die Registrateur van die Suid-Afrikaanse Mediese en Tandheelkundige Raad, Posbus 205, Pretoria, moet verwittig van enige adresverandering. Versuim hiervan beteken dat die *Tydskrif* nie afgelewer kan word nie. Dit het betrekking op lede wat oorsee gaan sowel as dié wat binne die Unie van adres verander.

* * *

Dr. T. J. Dry, 805 Medical Centre, Heerengracht, Cape Town, desires to intimate that his residential telephone number, as listed in the current telephone directory, is incorrect. The correct number is 7-1257.

Dr. T. J. Dry, Mediese Sentrum 805, Heerengracht, Kaapstad, vra dat kennis asseblief geneem moet word dat die huidige telefoongids sy huistelefoonnommer verkeerd aandui. Die regte nommer is 7-1257.

* * *

Dr. David Judes, M.B., B.Ch. (Rand), D.O., R.C.P. & S. (Eng.) is now practising as an ophthalmic surgeon at 304, Medical Centre, Jeppe Street, Johannesburg. Telephones: Rooms 22-6389, residence 44-1388.

* * *

Dr. Theunis Fichardt, M.D., M.Med. (Rad.), D.M.R.E., at the recent Graduation Ceremony of the University of Pretoria, obtained the D.Sc. degree with a thesis: 'The Value of Urethrocystography in the Roentgenological Investigation of the Normal and Pathological Anatomy of the Adult Male Urinary Bladder and Urethra.'

* * *

Medical Aid Tariff. In the new Tariff of Fees for Medical Aid Societies* the general practitioner's fee for a night visit (i.e. a call received and made between 7 p.m. and 7 a.m.) is £1 5s. The figure of £1 15s. which was printed in our Federal Council report* was inserted in error.

* Report (1957): S. Afr. Med. J., **31**, 388 (20 April).

* * *

South African Paediatric Association. The next meeting of the Cape Town Sub-group of this association will be held on Tuesday 7 May 1957 in the Lecture Theatre, Red Cross War Memorial Children's Hospital, Rondebosch, Cape, at 8.15 p.m. Dr. G. Sutin will give an address on Medical Experiences at the Children's Medical Center, Boston, Mass., (with emphasis on Cardiology and Poliomyelitis).

* * *

The Second International Congress for Social Medicine organized by the International Federation for Hygiene and Prophylactic Medicine, the Austrian Scientific Society for Social Medicine, and the Austrian Medical Chamber, will be held in Vienna, Austria from 30 May to 2 June 1957. The theme of the Congress is 'The University and Public Health'. The subjects for discussion will be the following: (1) Social Medicine and hygiene in the medical curriculum. (2) New approach to medical education. (3) Methods

of classification of population samples in medico-social studies. (4) The university hospital as a centre of clinical public health. There will be round-table discussions on the connotation of the university in connection with the following 4 topics: (*a*) Welfare of mother and child, family and school problems, clinical care of pregnant women and infants; (*b*) occupation, over-exertion, recreation, sport; (*c*) common diseases, medical statistics, health education, medical newspaper articles; (*d*) handicapped persons, geriatrics, rehabilitation, displaced persons, pension hunters, occupation of prematurely retired persons. The Secretarial Office for the Congress is Weihburggasse 10-12 ,Vienna 1.

* * *

NAPT Commonwealth Chest Conference. The next conference will be held on 1-4 July 1958 in the Royal Festival Hall, London. The provisional programme includes the following subjects: The world antituberculosis campaign; is it succeeding? The management of asthma in childhood. Ambulatory management of the tuberculous patient. Tuberculosis and leprosy. Thoracic surgery in respiratory tuberculosis. Tuberculosis and the psychiatrist. Lung cancer; prevention and treatment. The family and the patient with chest disease; how best can we help them to help themselves? Dust disease in miners. Tuberculosis medical and social services in British Overseas Territories.

An extensive exhibition will be held in the main foyer and terraces of the Royal Festival Hall. Visits to hospitals and clinics will be arranged as well as special clinical demonstrations, and also tours to places of general interest. A film demonstration will be held. Receptions and social engagements will be arranged.

Previous conferences have attracted thousands of delegates from 60 countries. The conference offers an opportunity for those interested in tuberculosis and diseases of the chest and heart, and other problems of preventive medicine, to meet workers in similar fields throughout the world, and to visit hospitals and clinics in Great Britain.

The membership fee is £5 5s. 0d., or 15s. per session, if the registration fee is received by 31 May 1958. The late fee is £6 6s. 0d. or 17s. 6d. per session. Delegates may obtain a copy of the transactions for an additional charge of 15s. 0d., but the volume will be printed only if sufficient orders are received. For further information write to the Secretary-General, NAPT, Tavistock House North, Tavistock Square, London W.C.1.

REVIEWS OF BOOKS : BOEKRESENSIES

THE POST-OPERATIVE RECOVERY ROOM

The Recovery Room. Immediate Postoperative Management. By Max S. Sadove, M.D. and James H. Cross, M.D. with contributions by 24 authorities. Pp. x + 597. Illustrated. $12·00. Philadelphia and London: W. B. Saunders Company. 1956.

Contents: Introduction. 1. An Administrator Looks at Intensive Therapy. 2. Principles of Recovery Room Management. 3. Management of Circulation, Shock, Respiration and Nutrition. 4. Neurosurgery. 5. Surgery of the Eye. 6. Surgery of the Ear, Nose and Throat. 7. Surgery of the Chest. 8. Laryngology and Bronchoesophagology. 9. Surgery of the Abdomen. 10. Surgery for Anorectal Disease. 11. Surgery of the Soft Tissues. 12. Orthopedic Surgery. 13. Urological Surgery. 14. Plastic Surgery. 15. The Treatment of Burns. 16. Oncological Surgery. 17. Obstetrics and Gynecology. 18. Vascular Surgery. 19. Pediatric Surgery. 20. Management of Medical Problems. 21. Nursing Care in the Recovery Room. Index.

The authors have produced a book which should be of immense value to hospital administrators, superintendents, matrons and heads of surgical and anaesthetic departments. Within its pages is found a most adequate description of the construction, equipment and the necessary medical and nursing personell required for the efficient functioning of the Recovery Room in a modern hospital.

The main emphasis is laid on the value to operation cases of specializ-d skill and special facilities in their treatment during the immediate post-operative period. But reference is also made to the saving in time of ward staff who are thereby released from those duties and are able to devote their full time and energies to the routine nursing requirements of the surgical ward; and the benefits which acrue to the other patients in a surgical ward, who are spared the inevitable disturbance of a patient recovering from anaesthesia, the constant visits of medical and nursing staff, switching of lights on and off, the rattle of trolleys conveying the requisites for intravenous therapy etc. are described and emphasized.

This volume contains valuable contributions from 24 well-known authorities, and is extremely well illustrated with photographs, diagrams and plans. There is a wealth of important clinical data on the post-operative treatment of both routine and complicated cases.

In a country where trained nursing personnel is at a premium, where modern hospitals are under construction, and the plans for future hospitals are still on the drawing boards, the contents of this timely publication are worthy of serious consideration by those responsible for our hospital services.

L.M.v.d.S.

CORRESPONDENCE : BRIEWERUBRIEK

PROPOSED AMENDMENT OF THE NURSING ACT, 1944

To the Editor: The Nursing Act of 1944 established a South African Nursing Association consisting of all registered nurses and midwives, irrespective of race and colour, for the purpose of raising the status, maintaining the integrity and promoting the interests of the nursing and midwifery professions. Provision was also made for the establishment of a South African Nursing Council to maintain discipline and professional status.

The Government, in conformity with its policy of *apartheid*, intends during this 1957 Parliamentary session to introduce an Amendment Bill to the Nursing Act to entrench racial discrimination in the nursing profession. The aim of this Bill is to:

1. Establish separate registers for different racial groups. Implied in this is the lowering of the status of the non-European nurse by the introduction of a separate syllabus and different uniform. We must assume that such differentiation will conform to the pattern set by the Bantu Education Act and the Separate University Education Bill.

2. Vest control of the South African Nursing Council in the hands of the Europeans by restricting membership of the Council to Europeans only.

3. Eliminate direct representation of non-Europeans on the South African Nursing Association. This may be done either:

(*a*) By establishing non-European standing committees (Coloured, Indian and African) 'to function under the direction of the Board (Europeans only) and to deal exclusively with non-European nursing matters'; or

(*b*) That the 'three groups (Coloured, Indian and African) should form their own separate nursing associations next to the statutory European body.' Thus, according to Dr. W. W. M. Eiselen of the Native Affairs Department, 'it would be possible to give European leadership and guidance to the Bantu nursing association in its various subdivisions or branches . . . by means of liaison officers appointed by the board of the statutory Nursing Association'.

The South African Nursing Council and the South African Nursing Association have both given evidence before a Select Committee of Parliament, appointed to investigate and draft the Amendment Bill. They both proposed racial discrimination in the nursing profession. The Council suggested the keeping of separate registers and the prescription of different syllabi because 'You cannot have one standard for a highly developed race and the

same for a lower developed race'. The Council further considered that the non-European nurses should not be eligible for election onto the Nursing Council, 'a body which exercises disciplinary powers over thousands of women in this country . . . (because) if such a nurse is able to sit in judgment on a European nurse who has transgressed, you would wreck the nursing service in South Africa'.[1]

The Board of the Nursing Association in turn recommended the establishment of a non-European standing committee, which has already been mentioned (3*a*). The Board was opposed to separate racial associations, as proposed by Dr. Eiselen a year later, because they feared that the statutory European Nursing Association would lose the privilege of membership of the International Council of Nurses. The Board also felt that the Europeans would then lose their controlling influence over the non-Europeans and they thought 'That the control of that (non-European) association will be seized by people who do not have the interests of nursing or the public at heart. They might be used for political purposes.' The Board claims to reflect South African nursing opinion on this matter. That this may be incorrect is shown by the fact that a referendum on this matter in 1950 was responded to by 28½% of the members of the Association entitled to vote, only 17% of the total electorate being in favour of racial discrimination. It is significant that the non-European groups were not informed by their branches of the fact that such a referendum was on the agenda of branch meetings and very few non-Europeans voted. This was particularly the case on the Witwatersrand. No reference appeared in the *South African Nursing Journal*, the official publication of the Association, to the referendum either before or after it was held. At the 1954 Biennial Conference of the Association the Board was criticized for this omission.

The non-European Discussion Group of the Witwatersrand Branch of the Nursing Association is opposed to the proposed Nursing Act Amendment Bill because it considers that the Bill will lower the professional status of the South African nurse and 'will have the effect of forcing South African nurses to contravene the International Code of Nursing Ethics, which all member organizations of the International Council of Nurses are expected to uphold, and to violate the best traditions of the nursing profession.' (Miss J. V. Kirchner, Assistant Secretary, Royal Australian Nursing Federation). We reject the non-European standing committee because we consider the non-European nurses of South Africa are competent to participate directly and fully in all the affairs of the nursing profession, and must not be restricted to 'non-European nursing matters', an indefinable entity. Dr. Eiselen's proposal of separate associations we also reject because we fear that we may thus lose our identity with the nursing profession, to be taken over by the Native Affairs Department, Coloured Affairs Department, etc. Such racial associations could hardly qualify South African nurses for membership of the International Council of Nurses, a situation we do not desire. In a memorandum prepared by us on the Bill we therefore respectfully requested that:

1. Concerning the register and the syllabus, the 1944 Nursing Act remain in force.

2. Concerning election to the Council and representation on the Board of the Association, there be no discrimination between the different racial groups, as was the position under the 1944 Nursing Act.

3. Provision be made in the new Bill for honouring and upholding the principle and the practice of equality in all the affairs of the South African Nursing Council and the South African Nursing Association. Discrimination should be made an offence.

The relationship between the medical and nursing professions is such that the doctors of South Africa cannot ignore the grave implications of the Bill soon to be presented to Parliament. The people in this country who depend on us for care and attention during illness will suffer grieviously as a consequence of the proposed lowering of the status and the education of the nurses. As a result of this the standard of medicine in the non-European hospitals will surely deteriorate. Further, the acute shortage of trained nurses in this country will be aggravated.

The strong possibility exists that non-European nurses may be removed from the control of the Nursing Association to be placed under that of non-nursing bodies, the Native Affairs Department and the Coloured Affairs Department, etc., as proposed by Dr. Eiselen in his evidence in 1956 before the Parliamentary Select Committee. The situation envisaged for the non-European nurses is similar to that original, hybrid Separate Universities Education Bill, which caused great anxiety to the medical profession. It is possible that concerted and determined action by the doctors may yet induce the Government to modify the Nursing Act Amendment Bill.

The South African Medical Association and Council may soon also be the victim of interference. Dr. Eiselen in his evidence before the Select Committee, when discussing the formation of a separate 'Bantu Association', said, 'I am aware of the fact that difficulties would be encountered if at this stage the Bantu nurses were deprived of membership and of voting rights on a mixed nursing association after these things had been granted to them. It would nevertheless be most inconsistent if on these grounds an anomaly was allowed to continue which exists in no other South African sphere of life—with the exception, I think, of the Medical Council and the Bar.'

The Board of the South African Nursing Association considers that because it has already committed the South African nurses to *apartheid* in the evidence before the Parliamentary Select Committee it is therefore unconstitutional for us to oppose the racial clauses. Because we contest this interpretation of the constitution and because we feel that it is essential that the terms of the Bill be made known, we have been forced to approach the nursing and medical profession and the public on this matter without the assistance of and in direct conflict with the Board of the Association. We nevertheless hope that our unofficial approach will not prevent the people from recognizing the urgency and the importance of the Bill. We further hope that our reasonable and democratic demands as outlined will be supported.

M. Ramusi (Mrs.),
Secretary, non-European Discussion Group of the Witwatersrand Branch of the South African Nursing Association.

1087 Dube Village,
Johannesburg
19 April 1957

1. Except where otherwise stated all quotations are from evidence given by representatives of the S.A.N.C. and the S.A.N.A. before the Parliamentary Select Committee.

‚MENEER' OF ‚DOKTER'

Aan die Redakteur: Vergun my die geleentheid om nog 'n stuiwer in die beurs te gooi. Ek het die afgelope weke se korrespondensie baie geniet.

Dr. (mnr.) Steenkamp meld in sy laaste brief[1] dat hy in die telefoongids en op sy naambordjie as Dokter' bekend staan. Nou ja, dit laat my terug dink aan 'n ondervinding in die verlede.

Ná ek klaargemaak het, het ek in 'n hospitaal gewerk waar die chirurge as ‚Mr.' aangespreek was. Ek het dikwels vergeet om dit te doen, omdat ek nie daaraan gewoond was nie. Juis by so 'n geleentheid het die chirurg my daarop attent gemaak dat hy 'n ‚Mr.' is. Toevallig ry ons 'n week later in dieselfde hysbak met ander mense saam. Ek spreek hom toe aan as ‚Mr.'. Toe ons onder kom vra hy my om hom liewers as ‚Dr.' aan te spreek wanneer ons in die publiek is!

T. B. de Bruyn

Cradockstraat 4
Steynsburg
19 April 1957

1. Steenkamp, W. (1957): S. Afr. T. Geneesk., **31**, 376.

ADVERTEERDERS SE AFRIKAANS

Aan die Redakteur: In die *Tydskrif* van 13 April, word aansoeke gevra vir, onder andere, ‚Een Deeltydse Borskwaaldokter' by die Oaktree-Hospitaal, Krugersdorp.

Wat se ding is 'n borskwaaldokter? Seer sekerlik tog nie 'n ‚Thoracic Surgeon' soos dit in die Engelse advertensie aangegee word nie?

Dit is hoog tyd dat Afrikaans reg gebruik word, ook in advertensies.

E. J. Marais

King Edward VIII-Hospitaal
Durban
17 April 1957